Industrial Disease Standards Panel

Comité des normes en matière de maladies professionnelles

REPORT TO THE WORKERS' COMPENSATION BOARD ON THE ONTARIO URANIUM MINING INDUSTRY

The Industrial Disease Standards Panel is a Schedule 1 Agency of the Government of Ontario attached to the Ministry of Labour. The function of the Panel, as defined in Section 86p of the Workers' Compensation Act of Ontario, is as follows:

(a) to investigate possible industrial diseases;

(b) to make findings as to whether a probable connection exists between a disease and an industrial process, trade or occupation in Ontario;

(c) to create, develop and revise criteria for the evaluation of claims respecting industrial diseases; and

(d) to advise on eligibility rules regarding compensation for claims respecting industrial diseases.

The Panel is required by statute to report its findings to the Workers' Compensation Board of Ontario.

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ON

THE ONTARIO URANIUM MINING INDUSTRY

Industrial Disease Standards Panel

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ON

THE ONTARIO URANIUM MINING INDUSTRY

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INDUSTRIAL DISEASE STANDARDS PANEL
IDSP REPORT NO. 6
FEBRUARY, 1989

Ontario Ministry of Labour
Ministère du Travail de l'Ontario

Industrial Disease Standards Panel
Comité des normes en matière de maladies professionnelles

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February 24, 1989

Dr. R.G. Elgie
Chairman
Workers' Compensation Board
2 Bloor Street East, 20th Floor
Toronto, Ontario
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Dear Dr. Elgie:

In accordance with Section 86p(10) of the Workers' Compensation Act, I am pleased to convey herewith IDSP Report No. 6 which addresses the issue of cancer in the Ontario uranium mining industry.

Yours sincerely,

J. Stefan Dupré
Chairman

JSD:jg

Ontario Ministry of Labour
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February 22, 1989

MEMORANDUM TO: WORKERS' COMPENSATION BOARD

FROM: INDUSTRIAL DISEASE STANDARDS PANEL

RE: REPORT ON ONTARIO URANIUM MINING INDUSTRY

1.0 THE BOARD REFERENCE

1.1 In a letter dated February 17, 1986, the Workers' Compensation Board requested that the Industrial Disease Standards Panel review the Board's guideline for adjudicating cases of lung cancer attributable to either radon or radon daughter exposure. The guideline was last revised by the Board on August 2, 1979 and appears in the Board's Manual of Policies and Procedures (Claims Adjudication Branch Procedures Manual, Document 33.13.13). The relevant sections of the guideline are reproduced in Appendix A of this report.

2.0 PANEL INVESTIGATIONS

2.1 The Board's letter of referral was received several months before the Panel was formally appointed. At its second meeting, the Panel received presentations and submissions from both labour and management parties of interest (Denison Mines Ltd.; U.S.W.A., September 9, 1986). By this time, a request to review issues of lung and stomach cancer among Ontario gold miners had also been received. As a consequence, the Panel appointed Professor H.S. Shannon of McMaster University to chair a Special Panel to conduct an epidemiological review of the entire Ontario mining industry beginning with gold mining. The Special Panel began its investigations into the uranium mining industry upon the completion of the Panel's gold mining report (Report on the Ontario Gold Mining Industry, IDSP Report No. 1, April, 1987).

2.2 The Report of the Special Panel on the Ontario Uranium Mining Industry is attached as Appendix B to this report. In it are described a number of undertakings including:

meetings with special advisers provided by labour and management parties of interest on historical conditions and practices in Ontario uranium mines:

- staff visits to Atomic Energy Control Board (AECB) offices in Elliot Lake and in Ottawa;
- a review of the epidemiological literature on health effects from radon daughter exposure; and
- an independent review and analysis of the epidemiological evidence on the mortality experience of Ontario uranium miners.

2.3 The complete evidentiary base used by the Panel in the preparation of this report is shown in Appendix C (a subset of which also appears in the Bibliography of the Report of the Special Panel in Appendix B). Appendix D contains a paper on the origins of radiobiological effects in uranium mining. Appendix E provides a comparison of low latency lung cancer cases among uranium miners in the Ham Commission Report with similar cases in the Report of the Special Panel.

3.0 PANEL FINDINGS

3.1 The Report of the Special Panel (RSP) reviews the overall and disease cause-specific mortality experience of Ontario uranium miners during the 1955-81 time period. There is a significant excess of deaths due to cancer of the trachea, bronchus and lung for all miners employed in an Ontario uranium "dusty job". "Dusty jobs" in mining are defined by the WCB occupation codes: 11-16, 21, 22, 25, 26 and 97 (See the RSP, pp.49-51, and Table 1 below).

TABLE 1

WORKERS' COMPENSATION BOARD OCCUPATION CODES FOR DUSTY MINING JOBS

WCB CODE	DEFINITION
11	Full time in dust exposure- Mill
12	Full time in dust exposure- Other surface
13	Full time in dust exposure- Shaft sinking
14	Full time in dust exposure- Other underground
15	Full time in dust exposure- Surface and underground
16	Full time in dust exposure- Open pit
21	Part time in dust exposure- Mill (including Mill in Open Pit)
22	Part time in dust exposure- Other surface
25	Part time in dust exposure- Surface and underground
26	Part time in dust exposure- Open pit
97	Dust exposure; specifics unknown

3.2 There is also a significant excess of lung cancer disease among those uranium miners who also had "other ore" exposures. This category has apparently been used as a coding

convenience by Board staff (in the updating of their Mining Master File) when the mined ore in question was unknown. Concerning this latter point, the RSP confirms that some uranium miners had some of their uranium mining experience labelled as "other ore" experience (pp.52-53).

3.3 The RSP finding of excess lung cancer disease among uranium miners has been repeatedly verified by many other local and international studies (see the RSP, Section 1.2, pp.31-39). As a consequence, the Panel makes the following finding:

FINDING 1: THE PANEL CONFIRMS THE EXISTENCE OF A SIGNIFICANT EXCESS OF MORTALITY FROM CANCER OF THE TRACHEA, BRONCHUS AND LUNG AMONG ONTARIO URANIUM MINERS IN THE DUSTY URANIUM JOBS THAT ARE DEFINED BY W.C.B. OCCUPATION CODES 11-16, 21, 22, 25, 26 and 97.

3.4 Among the remaining malignant neoplasms, the RSP found a slight, but non-significant, increase in stomach cancers especially among uranium miners with prior dusty gold mining experience (Tables 6 and 7, pp.69-70). However, as was found previously for Ontario gold miners, the epidemiological evidence for stomach cancer is biologically inconsistent with a finding of work-relatedness (p.53). There is no suggestion of an increase in lymphatic and hematopoietic tissue cancers, a category that includes leukaemia; nor is there any significant excess of kidney cancer.

FINDING 2: AT THIS TIME, THE PANEL DOES NOT FIND A PROBABLE CONNECTION BETWEEN ANY OTHER CANCERS AND OCCUPATIONAL GROUPS WITHIN THE ONTARIO URANIUM MINING INDUSTRY.

3.5 The Panel notes in passing that there is a significant excess of pneumoconioses (viz. silicosis) and related non-malignant respiratory diseases (viz. silicotuberculosis) which also have an occupational origin.

4.0 ELIGIBILITY RULES

4.1 In setting out to establish sound and fair eligibility rules for the adjudication of lung cancer claims for compensation among Ontario uranium miners, the Panel has kept in mind certain principles of clarity and simplicity that will allow both the adjudicator and claimant to understand clearly the rules against which an individual claim will, in the first instance, be adjudicated.

4.2 As a result of the Special Panel's findings (and that of other researchers) on gold mining and lung cancer and the Board's adoption of a compensation policy for this industrial

disease, there are in the current situation two groups of uranium miners with lung cancer who are potentially eligible for compensation. These are uranium miners with previous gold mining experience and uranium miners without previous gold mining experience. The Board's policy statement and guidelines for the compensation of gold miners with lung cancer calls for compensation where the following conditions are met:

- Proof of work in an Ontario gold mine;
- Medical evidence of a primary cancer of the trachea, bronchus or lung;
- Latency period of at least 15 years (between first employment in a 'dusty' occupation in a gold mine and the diagnosis of a primary lung cancer);
- Sufficient occupational exposure defined as one or more of the following conditions:

a) Chest x-ray rating of 4 or more AND a weighted dust index of 60 or more;

b) Chest x-ray rating of 4 or more AND first employment in an Ontario dusty gold mining occupation before the age of 30 years;

c) More than 5 years of dusty gold mining experience before 1945 AND first employment in an Ontario gold mine before the age of 30 years.

4.3 The first two conditions for the Board's gold miners' policy above are simply proof of diagnosis and of occupational work history. However, the third condition which stipulates latency period calls for at least 15 years. This is more stringent than the latency period of 10 years in the Board's current uranium guideline. The current evidence (p.56) shows that lung cancer risk from radon exposure begins to increase significantly 10 years following exposure. For this reason, the Panel recommends the retention of a 10 year latency period rule for lung cancer claims arising from uranium mining.

4.4 The Special Panel Report provides tables (Tables 22 and 23, pp.82-83) showing estimates of relative and attributable risk by various radiation exposure categories for the pure uranium miners (Table 22) and for the uranium miners with prior gold mining experience (Table 23). The tables also take into account the Panel's new recommendation concerning the manner in which the healthy worker effect (HWE) should be taken into account when evaluating epidemiological data (IDSP Report No. 3, July, 1988). Accordingly, the initial five years following first exposure have been excluded in the estimation of risk of mortality from lung cancer in order to eliminate any possible bias arising from the HWE. Note that the

relative risk coefficients shown in the footnotes to each table are as follows:

- Uranium miners 1.76% per WLM;
- Uranium and gold miners 1.63% per WLM.

Therefore, for the relative risk to reach a value of 2.0 or 200 (in percentage terms) would require, on average, 57 WLM (Working Level Months of radon exposure; see Appendix D for radiation definitions) of exposure for the pure uranium miners and 61 WLM for the uranium miners with previous gold mining experience.

4.5 At this point, the relative accuracy of exposure measurements must be taken into account. The Panel has a documented statement by Mr. A. Dory, Manager of the AECB's Uranium Mining Division, concerning the accuracy of the standard WLM estimates used by the Board (Dory, May 13, 1982). Exposure records are based on area monitoring alone and not on individual exposure records; and periodic reporting to the AECB of these area readings for each miner became a regulatory requirement only in 1968 (see RSP, Section 2, pp.40-45; and Appendix D). The approach used by the Board to determine an individual miner's radiation exposure has been based on the Working Level Table (Muller, 1983), a table which provides only two estimates (an 'average' and a 'high' estimate) of radon concentrations for each Ontario mine operating during each year in the period from 1955 to 1967 (and afterwards). Dory makes the following assertions concerning an individual miner's estimated WLM exposure:

- Prior to 1960, the uncertainty in WLM estimates is up to 1 order of magnitude (i.e. from 0.1 to 10 times the values shown);
- From 1960 to about 1970, the WLM uncertainty is of the order of plus or minus 200%;
- From 1970 on, the WLM uncertainty is of the order of plus or minus 100%;
- From about 1978 onwards, the uncertainty is within plus or minus 50%.

4.6 In another letter (Dory, April 1, 1982), Dory acknowledges the presence of other radioactive exposures (from gamma radiation and from thoron daughters) in some mining and mill operations in the province. Moreover, the Committee on the Biological Effects of Ionizing Radiation (BEIR IV, 1988) has stated that:

> "Dosimetric considerations suggest that the dose to the tracheo-bronchial epithelium from thoron progeny is, for an equal concentration of inhaled alpha energy, less by a factor of 3 than that due to the progeny of radon-222. The potential for lung cancer due to inhalation of thoron cannot be addressed directly, because the available epidemiological data are based almost exclusively on

exposure to radon-222 and its daughters." (BEIR IV, 1988. p.25)

4.7 The Panel notes, however, that estimates of exposure to radiation, although expressed in terms of the progeny of radon-222 in WLM, are in practice an index of total radiation exposure (including exposure to thoron progeny and to gamma radiation) and should be so regarded for compensation purposes. To derive estimates of total radiation exposure that incorporate additional data on thoron progeny (and/or gamma radiation) would be of no relevance to the placing of an individual worker in relation to the Panel's findings based on the RSP, which are derived from that component of a worker's exposure for which estimates of exposure in mines are available, namely exposure to radon progeny in WLM. As an example, consider a hypothetical worker with lung cancer whose cumulative exposure in WLM of radon progeny is recorded as 30 WLM. If it were possible to express his exposure to thorium and gamma radiation in terms of radon progeny WLM "equivalents", his total exposure might be estimated as 42 WLM which would include 30 WLM from radon progeny, 10 WLM from thoron progeny and 2 WLM from gamma radiation. However, it would clearly be an error now to attribute 42 WLM exposure to this worker, without at the same time performing similar calculations and corrections for all other uranium and gold plus uranium miners in the RSP. This would simply have the effect of reducing the estimated size of the lung cancer risk gradients (measured in percentage of relative risk per WLM) by about 29%. But these adjustments would not change the resulting risk estimates nor the conclusions drawn from the RSP, nor the placing of workers in relation to the probability that their lung cancer was due to radiation exposure. The Panel concludes that the dosage index used to determine eligibility for compensation, therefore, has to be on the same basis as that used by the RSP.

4.8 With this evidence in hand, the Panel has decided that the estimated difference between the doubling of risk of lung cancer for pure uranium miners and for uranium miners with prior gold mining experience (derived in Paragraph 4.4 above) is so small as not to merit the retention of any distinction between these two categories of uranium miners. Hereafter, in this report, a uranium miner will simply include any Ontario miner employed in a dusty uranium job defined by the W.C.B. Occupation Codes shown in Table 1 above.

4.9 With this simplification, the Panel considers that the appropriate starting point for new eligibility rules is to recommend compensation for any uranium miner who has met typical proofs of evidence regarding Ontario dusty uranium mining experience and a diagnosis of primary cancer of the trachea, bronchus and lung for which the latency period was at

least 10 years; and who has a cumulative WLM exposure of at least 40 WLM. This would provide some allowance for uncertainty in an individual's exposure; and for possible additional exposures to other radiation types.

ELIGIBILITY RULE 1: THAT CLAIMS ARISING FROM CANCERS OF THE TRACHEA, BRONCHUS AND LUNG AMONG ONTARIO URANIUM MINERS IN DUSTY OCCUPATIONS (W.C.B. OCCUPATION CODES 11-16, 21, 22, 25, 26 AND 97) AND MEETING THE FOLLOWING CRITERIA BE COMPENSATED:

1. **PROOF OF WORK IN A DUSTY ONTARIO URANIUM MINE;**
2. **MEDICAL EVIDENCE OF A PRIMARY CANCER OF THE TRACHEA, BRONCHUS OR LUNG;**
3. **LATENCY PERIOD OF AT LEAST 10 YEARS (BETWEEN FIRST EMPLOYMENT IN A 'DUSTY' OCCUPATION IN A URANIUM MINE AND THE DIAGNOSIS OF A PRIMARY LUNG CANCER);**
4. **SUFFICIENT OCCUPATIONAL EXPOSURE DEFINED AS A CUMULATIVE EXPOSURE TO RADON AND ITS PROGENY OF AT LEAST 40 WLM.**

4.10 However, given the magnitude of uncertainty in individual WLM estimates from 1955 even to the present, the Panel has decided to recommend a second eligibility rule to compensate any uranium miner whose cumulative WLM exposure falls between 20 and 40 WLMs, and who fulfills stipulated criteria:

ELIGIBILITY RULE 2: THAT CLAIMS ARISING FROM CANCERS OF THE TRACHEA, BRONCHUS AND LUNG AMONG ONTARIO URANIUM MINERS IN DUSTY OCCUPATIONS (W.C.B. OCCUPATION CODES 11-16, 21, 22, 25, 26 AND 97) MEETING CRITERIA 1, 2 AND 3 IN ELIGIBILITY RULE 1 ABOVE BE COMPENSATED IF THEY MEET <u>ONE</u> OF THE FOLLOWING CRITERIA:

1. **TOTAL WLM EXPOSURE IN THE TIME PERIOD 10 TO 14 YEARS (I.E. A 5 YEAR PERIOD) PRIOR TO DIAGNOSIS OF THE PRIMARY CANCER IS AT LEAST 20 WLM;**
   <u>OR</u>
2. **TOTAL WLM EXPOSURE IN THE TIME PERIOD 10 TO 14 YEARS (I.E. A 5 YEAR PERIOD) PRIOR TO DIAGNOSIS OF THE PRIMARY CANCER <u>PLUS</u> 0.5 (OR 50%) OF THE WLM EXPOSURE IN THE TIME PERIOD 15 OR MORE YEARS PRIOR TO THE**

DIAGNOSIS OF THE PRIMARY CANCER IS AT LEAST 20 WLM.

4.11 It should be noted that the second criterion of Eligibility Rule 2 is less stringent than the first criterion. Both are based on an appraisal of the RSP's modeling results (Tables 24, 25, 28 and 29 on p.84, 85, 87 and 88 respectively; and with explanatory text in Section 5.2, pp.54-57). What is material to both criteria is that the dose/response models show that the most important period in determining the risk for lung cancer is the period 10-14 years prior to observation or diagnosis. The second criterion recognizes that the next most important period is that 15+ years prior to observation (with about 50% of the importance of the 10-14 year period). As for the most immediate period of 5-9 years, it is not significant in the risk of producing lung cancer. The formulation employed in the second criterion has been used as well by the BEIR IV Committee in their modeling of the risk of lung cancer from radon exposure (RSP, pp.38-39; pp.54-57).

4.12 The Panel was concerned that the application of the above eligibility rules might exclude from compensation some legitimate claims of lung cancer among uranium miners. This could occur, for instance, among uranium miners with prior gold mining experience who contracted lung cancer before fulfilling the uranium latency criterion of 10 years. The Ham Commission (in Table C.4 of its Report) identified 19 cases of lung cancer among Ontario uranium miners with low latencies (of less than 10 years). Appendix E contains a comparison of these cases with similar cases from the RSP. It shows that twelve of fourteen higher exposure cases from the Ham Report were also in one or the other of the two Special Panel study cohorts (i.e. the GOLDBE or NOGOLDBE cohorts). Moreover, the overall degree of correspondence between the low latency cases in the Ham Report and the RSP is high (74%). For these reasons and others stated in Appendix E therefore, the Panel considers that the data in the Mining Master File which was used to create the Special Panel cohorts is a more complete and reliable list of Ontario uranium miners for epidemiological study purposes.

4.13 The Panel tested its Eligibility Rule 2 against the lung cancer cases in both study cohorts. For uranium miners with prior gold mining exposure (the GOLDBE cohort), Tables 2, 3 and 4 following show that 46 out of the 90 lung cancer cases (or 51%) are compensated according to this rule. There are no GOLDBE lung cancer cases fulfilling this criterion with less than 10 years of uranium dust latency (Table 2). Moreover, Table 3 shows that only 1 of these compensable cases had less than 15 years gold dust latency. Table 4 shows the gold and uranium latency characteristics of that portion of the GOLDBE cohort not compensated by the Panel's proposed uranium

TABLE 2

GOLDBE COHORT: LUNG CANCER MORTALITY
BY TIME SINCE FIRST EXPOSURE TO URANIUM DUST AND
THE PANEL'S PROPOSED URANIUM CRITERION

$W_2+0.5W_3$

TIME SINCE FIRST URANIUM DUST EXPOSURE (YRS.)	0-19			20-39			40+		
	OBS	EXP	SMR	OBS	EXP	SMR	OBS	EXP	SMR
0-5	2	1.76	1.14	0	0	-	0	0	-
5-9	10	4.34	2.30	0	0	-	0	0	-
10-14	5	4.57	1.09	4	1.88	2.13	5	1.87	2.68
15+	27	17.48	1.54	12	6.73	1.78	25	8.35	2.99
Total	44	28.16	1.56	16	8.61	1.86	30	10.21	2.94

TABLE 3

GOLDBE COHORT: LUNG CANCER MORTALITY
BY TIME SINCE FIRST EXPOSURE TO GOLD DUST AND
THE PANEL'S PROPOSED URANIUM CRITERION

$W_2+0.5W_3$

TIME SINCE FIRST GOLD DUST EXPOSURE (YRS.)	0-19			20-39			40+		
	OBS	EXP	SMR	OBS	EXP	SMR	OBS	EXP	SMR
0-5	0	0.03	0.00	0	0	-	0	0	-
5-9	0	0.25	0.00	0	0	-	0	0	-
10-14	1	0.84	1.20	1	0.08	12.64	0	0.04	0.00
15-24	8	5.74	1.39	3	1.53	1.96	2	1.70	1.17
25+	35	21.30	1.64	12	7.00	1.71	28	8.47	3.31
Total	44	28.16	1.56	16	8.61	1.86	30	10.21	2.94

TABLE 4

GOLDBE COHORT: LUNG CANCER MORTALITY
BY TIME SINCE FIRST EXPOSURE TO URANIUM DUST AND
TIME SINCE FIRST EXPOSURE TO GOLD DUST(FOR URANIUM CRITERION = 0-19 WLM)

TIME SINCE FIRST URANIUM DUST EXPOSURE (YRS.)	TIME SINCE FIRST GOLD DUST EXPOSURE														
	0-4			5-9			10-14			15-25			25+		
	OBS	EXP	SMR	OBS	EXP	SMR	OBS	EXP	SMR	OBS	EXP	SMR	OBS	EXP	SMR
0-5	0	0.03	0.00	0	0.11	0.00	0	0.18	0.00	0	0.67	0.00	2	0.76	2.63
5-9	0	0	-	0	0.14	0.00	1	0.41	2.44	3	1.36	2.21	6	2.44	2.46
10-14	0	0	-	0	0	-	0	0.24	0.00	1	1.24	0.81	4	3.09	1.30
15+	0	0	-	0	0	-	0	0	-	4	2.48	1.61	23	15.0	1.53

TABLE 5

GOLDBE COHORT: LUNG CANCER CASES (WHO DO NOT MEET THE PANEL'S URANIUM CRITERION) IN RELATION TO WCB GOLD MINING COMPENSATION CRITERIA

TIME SINCE FIRST URANIUM DUST EXPOSURE YRS.	LUNG CANCER CASES OBS O	EXP E	EXCESS O-E	CASES MEETING CRITERIA FOR WCB GOLD MINING COMPENSATION
0-5	2	1.76	0.24	1
5-9	10	4.34	5.66	5
10-14	5	4.57	0.43	2
15+	27	17.48	9.52	8
Total	44	28.15	15.85	16

TABLE 6

NOGOLDBE COHORT: LUNG CANCER MORTALITY BY TIME SINCE FIRST EXPOSURE TO URANIUM DUST AND THE PANEL'S PROPOSED URANIUM CRITERION

$W_2+0.5W_3$

TIME SINCE FIRST URANIUM DUST EXPOSURE (YRS.)	0-19 OBS	EXP	SMR	20-39 OBS	EXP	SMR	40+ OBS	EXP	SMR
0-5	2	1.35	1.48	0	0	-	0	0	-
5-9	6	2.86	2.10	0	0.00	0.00	0	0.00	0.00
10-14	6	3.76	1.60	3	1.13	2.65	1	1.13	0.89
15+	25	18.42	1.36	6	2.91	2.06	17	4.19	4.05
Total	39	26.39	1.48	9	4.04	2.23	18	5.32	3.38

criterion (some 44 cases). Table 5 shows that 16 out of these 44 cases would be compensated according to the Board's current gold mining eligibility rules (see Section 4.2 above). In total, therefore, 62 out of 90 lung cancer cases in the GOLDBE cohort or 69% would be compensated according either to the Board's current gold mining rules or the Panel's proposed eligibility rules. For the pure uranium miners (the NOGOLDBE cohort), Table 6 shows that 27 out of 66 (or 41%) of the lung cancer cases would be compensated according to Eligibility Rule 2.

5.0 CASE BY CASE ADJUDICATION

5.1 Despite the assurances provided by the analyses described above in Sections 4.12 and 4.13 (and in Appendix E), the Panel is concerned that there might still remain legitimate cases of lung cancer among either gold miners or uranium miners (with or without previous gold mining experience) which fall just short of qualifying for compensation under either the Board's gold mining eligibility rules or the proposed eligibility rules for uranium miners in this report.

5.2 For this reason, the Panel urges the Board to adhere to the following recommendation when adjudicating claims on a case by case basis.

RECOMMENDATION 1: **IN ADJUDICATING ANY CLAIM FOR COMPENSATION FOR CANCERS OF THE TRACHEA, BRONCHUS AND LUNG AMONG ONTARIO GOLD OR URANIUM MINERS, THAT THE BOARD TAKE FULL COGNISCENCE OF THE FOLLOWING CONSIDERATIONS:**

1. **THE RANGE OF ADDITIONAL EVIDENCE IDENTIFIED BY THE PANEL IN ITS CRITERIA OF CASE EVALUATION (SECTION 4.0, REPORT ON THE ONTARIO GOLD MINING INDUSTRY, IDSP REPORT NO. 1, APRIL, 1987).**

2. **THE MANY INACCURACIES WHOSE PRESENCE IS ACKNOWLEDGED IN THE AVAILABLE ESTIMATES OF ANY INDIVIDUAL URANIUM MINER'S RADIATION EXPOSURES AS A RESULT OF:**
   - **THE USE OF RADIATION READINGS BASED ON AREA SAMPLING RATHER THAN INDIVIDUAL RECORDS;**
   - **THE ABSENCE OF RECORDS ON KNOWN EXPOSURES TO OTHER RADIATION TYPES SUCH AS GAMMA AND THORIUM;**

- **THE FACT THAT NO RECORDS ON RADIATION EXPOSURES IN EITHER SURFACE OR MILL WORK ARE KEPT;**
- **THE FACT OF EXPOSURE TO OTHER UNIDENTIFIED CARCINOGENIC SUBSTANCES FOR URANIUM MINERS WITH PRIOR GOLD MINING EXPERIENCE (IN THE PANEL'S GOLD MINING REPORT, POSSIBLE CARCINOGENS INCLUDED RADON, SILICA, AND ARSENIC).**

3. **THE PROVISIONS STIPULATED IN SECTION 3(4) OF THE ONTARIO WORKERS' COMPENSATION ACT WHEN WEIGHING ALL OF THE ABOVE ITEMS OF EVIDENCE.**

5.3 Beyond this recommendation for the appropriate consideration by the Board of lung cancer claims among hard rock miners in this province who may have been at significant occupational risk of contracting lung cancer and whose employment histories may not be encompassed by current and proposed eligibility rules, the Panel proposes to conduct its own study in order to determine the characteristics of these individuals. For example, they may have had a history of gold mining followed by a small amount of uranium mining exposure neither of which was sufficient to qualify them under either the Board's gold or the Panel's proposed uranium eligibility rules. In addition, the possible roles of silica, radon exposure (in non-uranium mines), arsenic and diesel emissions as causal agents in the production of their lung cancer will be explored. It is the Panel's intention to describe the occupational characteristics of the groups of miners who were at significant risk of excess lung cancer according to the combinations of ores mined, latency periods, exposure categories and other factors.

Ontario Ministry of Labour
Ministère du Travail de l'Ontario

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Comité des normes en matière de maladies professionnelles

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February 22, 1989

MEMORANDUM TO: **THE WORKERS' COMPENSATION BOARD**

FROM: **LINDA JOLLEY, JEAN GAGNON AND JOHN CHONG, MEMBERS OF THE INDUSTRIAL DISEASE STANDARDS PANEL**

RE: **DISSENT FROM THE MAJORITY REPORT ON THE ONTARIO URANIUM MINING INDUSTRY**

We concur with all the items in Sections 1.0, 2.0 and 3.0 of the Majority Report of the Panel. This dissent addresses Sections 4.0 and 5.0 of that Report.

In Paragraph 4.5, the Panel addresses the relative accuracy of the exposure measurements in the uranium mines. Individual miner's exposure limits before 1968 are based on area samples only, and while the Panel recognizes the magnitude of error in determining an individual miner's estimated WLM exposure as asserted in the documented statement by Mr. A. Dory, AECB, the potential inaccuracies have not been properly addressed in the establishment of the exposure requirement of both Eligibility Rule 1 and 2.

The Report of the Special Panel on the Ontario Uranium Mining Industry uses the average exposure level for the mine as an estimate of the typical exposure incurred by each individual. However, collective agreements from late 1958 until the present, contained job posting provisions based on seniority and ability that restricted miners to specific jobs that they bid on and did not allow them to move between a variety of jobs within the year. While the results in the Special Panel Report are consistent with a dose-response relationship based on these average mine exposures, an individual miner's exposure may in reality be much higher since he worked on a specific job, like driller or slushermen, with a much higher exposure which must be considered for compensation.

In addition, surface and mill workers have been recognized as having radon exposure on their jobs, and yet the Mining Master File records zero exposure.

Recommending consideration of such inaccuracies on a case by case basis does not address this concern sufficiently, since case by case adjudication takes place only on cases which just fall short of qualifying for compensation. Using Dory's assertion that uncertainty in individual measurements before 1960 is up to 1 order of magnitude would mean that an average WLM exposure of 15 WLM could be as high as 150 WLM, and if uncertainty between 1960 and 1970 is of the order of plus or minus 200%, would mean that an average WLM exposure of 15 WLM occurring before 1970 could be as high as 45 WLM, and yet neither case would be considered to fall just short of the eligibility rule. Nor would zero exposure for a surface or mill worker be considered in such case by case adjudication.

In Paragraph 4.6, while the radon exposure levels reflect an index of total radiation exposure among those miners covered in the Special Panel's Report, the eligibility rules will apply to all uranium miners and mill workers in this province. Agnew Lake is a relatively new mine which has not had sufficient latency to produce the cancer that may well develop in the future. But Agnew Lake has a higher ratio between thoron and radon daughters than in Elliot Lake from which much of the experience in the Special Panel's Report is drawn.

As well, until 1966, Rio Tinto Dow operated a thorium plant in Elliot Lake, the main purpose of which was the production of thorium and not uranium. But those workers would have been exposed to significant levels of thoron progeny which cannot be expressed by using radon exposure as an index for total radiation exposure.

In Paragraph 4.9, requiring only latency in uranium mining in Eligibility Rules 1 and 2, means that the period of actual radon exposure or exposure to other potential lung carcinogens like arsenic or silica experienced in gold mining prior to uranium would not be considered. As the exposure criteria are stated, however, radon exposure in those gold mines could be considered in estimating total exposure, which seems rather inconsistent. A miner with ten years of experience in gold, who then spends seven years in a uranium mine would not meet either the 15 year latency requirement of the gold mining Eligibility Rule or the proposed ten year latency requirement in the uranium mining Eligibility Rules 1 or 2, and yet there is actually 17 years of potential exposure.

While it is clear that there is a latency period between first exposure and actual death from lung cancer which may be in the order of ten years, it is essential that eligibility rules if applied consider all exposure, and if costs must be shared between classes of employers then so be it, although all mining comes under class 5 of the Workers' Compensation Act, Schedule 1.

The translation of scientific evidence concerning occupational exposures and diseases in the community into public policy pertaining

to compensation of victims of occupationally-related illness must go beyond the simple extension of risk categories into eligibility rules. Consideration of historical and social contexts in which compensation policy is cast and the burden of proof upon individual claimants to obtain just and fair settlements must be included in an overall assessment of the evidence.

Epidemiology is able to identify a probable connection between radioactive exposures and lung cancer. However, it cannot identify those individual miners whose lung cancer is occupationally related and those whose lung cancer is not. Nor should compensation be limited to only those miners who fall within a statistical excess identified by latency and average WLM exposure, especially given the huge inaccuracies in individual exposure extrapolations from average mine readings and the failure to recognize latency before entry into uranium mining.

Nor do we believe that a case by case assessment for those who just fall short of the Eligibility Rules proposed by the majority report addresses the problem that workers have in bearing the burden of proof. Workers are then faced with the exercise of attempting to make sense out of infrequent area sampling, varying geology, mining methods, differing ventilation practices, overtime, etc. that according to the AECB itself, influences the uncertainty of each individual's exposure. Workers must bear not only the burden of their diseases, but the burden of proving their work relatedness, as well.

Since we believe that a probable connection between lung cancer and uranium mining and milling has been demonstrated, it is appropriate for the Workers' Compensation Board to use Section 122(9) of the Workers' Compensation Act for the purposes of compensating those workers.

For these reasons, we as Panel Members recommend:

ELIGIBILITY RULE: THE BOARD SHOULD ENTER CANCER OF THE LUNG INTO SCHEDULE 3 OF THE WORKERS' COMPENSATION ACT, AND THAT CANCER OF THE LUNG SHALL BE DEEMED TO HAVE BEEN DUE TO THE NATURE OF URANIUM MINING, MILLING AND SURFACE WORK AND URANIUM MINING, MILLING AND SURFACE WORK WITH PRIOR GOLD MINING EXPOSURE, UNLESS THE CONTRARY IS PROVED.

APPENDIX A

WORKERS' COMPENSATION BOARD

GUIDELINES FOR ADJUDICATION

LUNG CANCER - RADON AND

RADON DAUGHTERS

GUIDELINES FOR ADJUDICATION

LUNG CANCER - RADON AND

RADON DAUGHTERS

1. Primary lung cancer caused by exposure to Radon Gas and Radon Daughters in industry shall be accepted as an industrial disease under Section 122, Section 1(1) (N) of the Act and Schedule 3- Item 9.

2. Based on Ontario and other experience lung cancer claims in which the latency period is at least 10 years shall be considered using the following factors:

   2.1 Duration of exposure
   2.2 Exposure Intensity
   2.3 Smoking history
   2.4 Geographical location of exposure
   2.5 Age when lung cancer first appears
   2.6 Year of entry into mining
   2.7 Age at start of exposure
   2.8 Previous underground exposure in non-uranium mining

3. Claims which fail to meet the criteria in Section 2, shall be considered on their own merits having regard to all factors. The benefit of reasonable doubt applies.

APPROVED BY THE BOARD

August 2, 1979

APPENDIX B

REPORT OF THE SPECIAL PANEL

ON THE

ONTARIO URANIUM MINING INDUSTRY

REPORT OF THE SPECIAL PANEL ON THE

ONTARIO URANIUM MINING INDUSTRY

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FOREWORD

The Special Panel on the Ontario Uranium Mining Industry was Chaired by Professor Harry S. Shannon of McMaster University, Hamilton, Ontario. Dr. James G. Heller, the Panel's Executive Administrator, co-authored the Report of the Special Panel with Professor Shannon and directed the support work of Panel staff. Staff contributors to the Report include: John Gerritsen, Sheldon Davis (contract programmer), Annette Matuszek (Ministry of Labour programmer), Noella Martin, Paul Gallina and Gaylene Pron. The manuscript was typed by Madeleine Dennison and Donna Cowan.

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EXECUTIVE SUMMARY

1. The aim of this report was to identify any significant excesses of mortality among uranium miners in Ontario and to rank cases for those diseases found to be significantly increased according to the likelihood that they were occcupationally related.

2. A review of the literature showed that uranium miners and others exposed to radon daughters had been consistently found to experience an increase in lung cancer mortality. However, no other cause of death was found to be consistently increased in the various studies.

3. A recent report by the Committee on the Biological Effects of Ionizing Radiation (known as BEIR IV) examined the effects of alpha emitters, including radon and its progeny, and derived a relationship for the risk of lung cancer following radon exposure. It was expressed in terms of the age at risk and the prior pattern of radon exposure.

4. Panel staff met with staff of the AECB (Atomic Energy Control Board) to learn about exposures and exposure measurements in uranium mines, and the concurrence between AECB records and those contained in the Mining Master File at the Workers' Compensation Board, which were used for this study.

5. Panel members met with two advisers, who described past conditions and practices in Ontario uranium mines. They pointed out that conditions have improved gradually since the mines began operation in 1955.

6. Death certificate data on cause of death for all lung cancer cases were validated using the Ontario Cancer Registry for corroborating information. The diagnosis of lung cancer on death certificates was found to be validated in 97% of cases, a higher rate than that for the Ontario male population.

7. Since many uranium miners had previously worked in gold mines, for which there has been demonstrated an occupational lung cancer risk, the information collected from a Panel report on gold miners was employed to estimate the effect on lung cancer risk from prior gold dust exposure.

8. Further analyses were conducted using a cohort defined as follows: men were included in the study if they had attended a miner's chest x-ray examination at any time between January 1, 1955 and December 31, 1981 at which time the miner reported at least one half month's employment in an Ontario uranium dusty job and current employment in an Ontario uranium mine.

9. Various subdivisions of the cohort were made. For example, person years were divided according to whether the miner had any previous gold mining experience or not.

10. A statistically significant increase in lung cancer was found. In addition, there was a five-fold increase in deaths due to chronic interstitial pneumonia, a category that includes pneumoconiosis - a known disease of miners. Further, the number of deaths due to "accidents, poisoning, and violence" was double that expected. No other specific cause of death was significantly increased.

11. Both the relative and attributable risk of lung cancer increased with increases in cumulative exposure to radon.

12. A statistical modelling approach was used to incorporate other factors, as well as to allow for any gold mining experience. The technique used is known as Poisson regression.

13. The preferred model adopted to describe the risk of lung cancer among uranium miners with prior gold mining experience was based on the cumulative weighted dust exposure in gold mining, the age at risk, and the cumulative exposure to radon in two periods, 10-14 years before observation and 15+ years before observation. Other models are provided to describe lung cancer risk among uranium miners with no prior gold mining experience.

14. A ranking of the likelihood that any case of lung cancer was caused by occupational exposures in Ontario uranium mines can be derived from the models indicated above.

SECTION 1

HISTORICAL CONTEXT

1.1 INTRODUCTION

The Special Panel was requested to provide an epidemiological review of mortality from all causes among uranium miners in Ontario and, for any significant excess of deaths from any cause, to rank the cases according to the likelihood that the disease was occupationally related. The agent of interest is presumed to be radon and its progeny. The need for this review followed a report on Ontario miners containing the following major findings (Muller, 1983):

1. an increase in lung cancer in gold miners;
2. an increase in stomach cancer in gold miners;
3. an increase in lung cancer in uranium miners.

A previous report of the panel (IDSP, 1987) addressed the issues of lung and stomach cancer among Ontario miners with only gold mining experience. A further report (Muller, 1987, 1988) re-examined the data for Ontario uranium mining. This review provides further epidemiological analyses of uranium mining, and, in particular, examines the effects of prior gold mining experience among uranium miners on the risk of increased lung cancer. The statistical analyses required to model the risk from radon progeny of lung cancer among uranium miners are necessarily complex, but an effort to explain the approaches in lay terms is provided.

The remainder of this chapter is devoted to a review of the epidemiological literature on health effects from radon exposure.

1.2 LITERATURE REVIEW

1.2.1 Introduction

As will be seen below, several previous studies of uranium miners have found an increased risk of lung cancer. The effect is believed to be due to the presence of radon gas and its decay product, radon daughters. This results in environmental radioactivity in the form of alpha particles. From the standpoint of health effects, the essential difference between alpha emission and gamma radiation (eg. x-rays) is that the energy of the alpha particles is absorbed and retained by any material into which the particles penetrate. Thus, alpha radiation will be absorbed by the skin - or by the lining of the lungs if the particles are inhaled - while gamma radiation will pass through the body. This means that the likely effect of alpha absorption would be damage to the lungs (or perhaps the skin). Further details on radiobiological effects are provided in Appendix A to this Report.

1.2.2 International Review Committees On Radiation

Extensive research has been conducted on the relationships between radiation exposures and human health risks. Reports published by scientific committees established by the United Nations and by the U.S. National Academy of Sciences have been major sources of information on radiation effects. The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), established in 1955 by the United Nations, was charged with two major tasks regarding the effects of human exposures to radiation: to monitor radiation exposures throughout the world; and to produce reports summarizing current worldwide scientific opinion and information on radiation effects. The other major committee, the Committee on the Biological Effects of Ionizing Radiation (BEIR), was established in 1964 by the National Academy of Sciences at the request of the Environmental Protection Agency and the Federal Radiation Council as an advisory body on all radiation matters affecting human health.

The BEIR committee was charged with reviewing and evaluating all the available scientific evidence on radiation exposures and effects. It has published four major reports known as BEIR I,II, III and IV. These reports constitute major reviews of scientific information on the field of radiocarcinogenesis. The BEIR IV report, issued in January 1988, was devoted to the health risks of alpha-emitters, with the primary focus on radon and radon daughters. The committee had access to the actual data from four published studies of uranium miners and derived an equation to describe the risk of lung cancer following exposure to radon progeny. A review of these and other important studies and a discussion of factors that may affect risk follows.

1.2.3 Epidemiological Studies of Miners Exposed to Radon

· Uranium Mining Cohorts

Epidemiologic studies of uranium miners, concerned primarily with the risk of lung cancer following exposure to alpha radiation from short lived radon daughters, come from three countries (Czechoslovakia, Canada and the United States). Among Czechoslovakian miners from the Joachimsthal area, increased lung cancer mortality was reported in studies involving two different follow-up periods (Sevc 1976, Kunz 1979). The Colorado Plateau (four states) cohort, followed since 1950, has had the longest period of observation. The first report of increased lung cancer mortality was published in 1962 by Archer. Further reports with extended follow-up (Wagoner, 1963, 1964, 1965; Lundin, 1969, 1971, 1979; Waxweiler, 1981, 1983; Whittemore, 1983; Hornung, 1985,1987) have also described increased lung cancer mortality. A study of New Mexico uranium miners has also been reported (see Samet, 1984). Studies in Canada have also reported excess lung cancer in three cohorts: in Ontario (Muller, 1983, 1987, 1988); in Saskatchewan (Howe, 1986); and in the Northwest Territories (Howe, 1987). Three studies from Ontario have reported different follow-up periods: Hewitt, 1976 (from 1955 to 1974); Muller, 1983 (from

1955 to 1976); and Muller, 1987 (from 1955 to 1981). Further details will be given below.

Non-Uranium Mining Cohorts

Mortality studies showing increased lung cancer risk with occupational exposure to radon daughters have also been reported in other types of miners - iron, zinc, tin and fluorspar. Survey results (Duggan, 1970) that reported radon levels to be higher in tin and hematite mines than in coal mines, for which increased risks of lung cancer have not been reported, suggest perhaps that exposure to radon and not underground mining per se is the contributing factor although other environmental contaminants may also be present. Studies on iron miners have been reported from several countries: for example, in the Malmberget (Radford, 1984) and Grangesberg (Edling, 1982, 1983) mining regions of Sweden; and in the Cumberland mining region of Britain (Boyd, 1970). Of these different mining groups, the Malmberget miners of Sweden have been investigated the most extensively. They were followed from 1951 to 1976. Although a similar period of observation (1957-77) was employed for the Grangesberg iron miners, the cohort itself was not as well defined. Minnesota iron ore miners did not show an increase in lung cancer (Lawler, 1985), although the authors were uncertain if this difference from other cohorts might have been because of low radon levels, strict enforcement of smoking prohibition, silicosis control or absence of diesel use underground. Mortality studies of tin miners in Cornwall have also reported increased lung cancer mortality (Fox, 1981).

Excess lung cancer was reported for fluorspar miners in Newfoundland as early as 1950 (Morrison, 1981) and the measurement of high levels of radon after 1959 suggested this was the cause. Mortality studies on this cohort involved follow-up of underground miners from 1933 to 1961 (deVilliers, 1964, 1971) and an extended follow-up to 1977 (Morrison, 1981). Both studies reported significantly increased lung cancer mortality.

1.2.4 Estimation of Cancer Risk

Given the results from these studies, a causal relation between lung cancer and exposure to radon is uncontested (BEIR IV, 1988; UNSCEAR, 1982) and has been acknowledged for many years. However, there is disagreement on other aspects of radon carcinogenesis. Interpreting and comparing results from epidemiologic studies conducted on different uranium and non-uranium cohorts involving variable study periods, mortality updates and analytic techniques, is a formidable task. Indeed, this is why the BEIR IV Committee obtained the raw data from the uranium miners studies, so that the same analytical methods could be applied to each thus enabling direct comparisons across cohorts.

Attempts to describe the relationship between exposure to radon and lung cancer are complicated by the lack of agreement on the appropriate underlying biological model of carcinogenesis. Various

ones have been proposed, such as the one-hit model or multistage model. However, because the biological mechanisms are not known, descriptions are commonly empirically based.

The simple (traditional) approach is for the mortality rates of the cohort to be compared with the mortality rates from a reference population, usually provincial or national. There are two potential problems associated with this technique: there may be differences between the cohort and the reference group; and even with large numbers of deaths in the cohort, after stratifying for various confounders (age, calendar year, etc.), the resultant small number of deaths often results in unstable estimates of risk for comparison. To address these difficulties, the use of statistical models has become increasingly popular. However, their advantages must be balanced against their complexity. Decisions on models are also complicated by a need to understand how other relevant variables may influence or modify cancer risk, and hence the probability that an individual cancer is work-related. The use of modelling techniques to estimate risks and dose-response relationships is examined in the following section.

· Factors To Consider in Modelling

· · Dose and Dose Rate Effects

Dose, generally measured as cumulative exposure in radon carcinogenesis, is a function of duration and intensity of exposure. As discussed above, the standard unit of exposure to radon daughters is the working level month (WLM). Low exposures over prolonged periods of time or high exposures over short periods of time can lead to the same cumulative WLM. This will not necessarily lead to the same risk since it is possible that the risk per unit exposure may vary depending on the rate at which the total dose was accumulated. That lower rates of radiation exposures may be more harmful is consistent with the principles of radiobiology. Levels of radiation that kill cells outright are not believed to be as important for carcinogenesis as levels of radiation that result in lesser damage to cells. The lesser damage involving errors in chromosomal DNA could result in cancer (and also genetic anomalies) in subsequent cellular proliferation (Pochin, 1980). This does not occur when cells are killed, since they then cannot mutate to become cancerous and the normal process of cell division would be expected to replace the lost cells.

· · Latency and Lag Periods

The latent period for carcinogenesis refers to the length of time from first exposure to the diagnosis of or death from the disease of interest. For solid tumours, this period is usually lengthy and variable (Armenian, 1974). This period may be affected by several modifying factors: concentration of exposure, age at time of first exposure, and other exposures. Because exposure usually occurs over extended periods of time, and because the preclinical changes in tumorigenesis are hidden, it becomes exceedingly difficult to estimate

the period of latency. In mortality studies, the latent period is defined to be the time from initial exposure to death.

It seems biologically unreasonable to count exposures in the period immediately preceding death since they will not have contributed to the carcinogenic process. A standard technique used in the analysis of cohort studies to compensate for this fact is to "lag" the exposures, i.e. to ignore exposures accumulated in a selected time period immediately preceding death or observation. However, estimates of cancer risk coefficients have been shown to vary with the choice of lag period (Hornung, 1985), although five years may be a reasonable minimum when mortality from lung cancer is examined.

· · Temporal Factors

Risk models can also incorporate temporal factors that modify the relationship between exposure and disease. Temporal factors are determinants or modifiers of disease that vary with age at risk and/or time. The temporal factors usually considered in epidemiological studies are: calendar date of first exposure; age at first exposure; age at risk or observation (usually age at death); time since initial or final exposure; and duration of exposure. All of these have been shown to influence or modify dose-response relationships in studies of various cancer types (Lundin, 1979; BEIR III, 1980; Day, 1980). However, the interpretation of the effects of these variables is complicated by the fact that they are inter-correlated. The comments below on the relationship derived by the BEIR Committee will provide more detail.

1.2.5 Review of Epidemiologic Studies on Miners Exposed to Radon

As noted earlier, there have been five main historical cohort studies of uranium miners in Czechoslovakia, United States, Saskatchewan, the Northwest Territories and Ontario.

The reports were based on historical cohort studies. As the name implies, these use records to identify workers at some point in the past from some particular company, job or occupation. Both personal and exposure records are abstracted to try to determine both quantitative and qualitative levels of exposure to environmental contaminants. Deaths among the workers are determined, and the cause and date of death established. Finally, the analysis consists of comparing mortality among the workers with some suitable reference group (commonly the general population) to determine whether mortality from some cause or causes is higher than would be expected. Allowance can be taken of the extent of exposure as well as other factors predicting disease risks.

Apart from the aforementioned studies, additional reports from France and New Mexico have also been reported (Tirmarche, 1985; Samet, 1984). The French report has inherent limitations, including the lack of availability of mortality data, the apparent incorrect computation of

person years, and omission of exposure concentrations and duration which prevent the drawing of conclusions. The only epidemiological results reported were for lung-cancer mortality: 36 observed cases compared with 18.8 expected among the entire cohort.

The New Mexico reports (Samet, 1984; Morgan and Samet, 1986) are cohort studies of lung cancer mortality in approximately 4,000 underground uranium miners in this state. A comprehensive report has not yet been published. However, a case control study involving 65 lung cancer cases and 230 age-matched controls shows increased risk for all cumulative exposures to radon progeny of 100 WLM or greater. With exclusion of subjects with exposures above 1,000 WLM, the estimated excess relative risk was 1.05 percent per WLM. The data were consistent with a multiplicative interaction between cigarette smoking and exposure to radon progeny.

Howe et al. (1986) reported a cohort study of 8,487 workers at a uranium mine in Saskatchewan, Canada. All men who had worked between 1948 and 1980 were included, although some had to be excluded because no birth year was recorded, job histories were incomplete, recorded birth year was in error or they had worked for the company elsewhere (so presumably had other radiation exposures). Altogether, the exclusions came to over 20% of all male workers. Sixty-five (65) lung cancer deaths occurred, nearly double the number expected. A strong dose-response relationship by cumulative WLM was observed. This was true both for attributable risk and for relative risk. The authors found that the "simple linear model gave a very good fit to the data", and lagging dose by five years led to an estimate of 3.5% increase in lung cancer relative risk per WLM. The authors noted that silica exposures were always very low and diesel machinery had not been used underground.

Several studies have been reported of uranium miners in Czechoslovakia (Sevc, 1976; Sevc, 1988). Unfortunately, very little information is provided on the methodology of these studies. Thus it is impossible to determine the presence and effects of possible flaws in the study on reported results. The most recent (1988) report includes six groups, two of which were not uranium miners (iron and shale clay) but were exposed to radon. The first two groups, who began employment in 1948-1952 and 1953-1957 with mean exposures of 303 and 134 WLM, respectively, experienced 484 lung cancer deaths, roughly five times the number expected. In contrast, the second two groups, who began uranium mining in 1968-1972 (and were followed for a maximum of only 13 years) and 1973-1975 (maximum follow up 8 years) had mean exposures of only 6.1 and 3.2 WLM, respectively. Four lung cancer deaths occurred compared with 3.1 expected. The other two cohorts had intermediate exposures (40 WLM and 25 WLM) and experienced intermediate relative risks. Many of the calculations presented estimated attributable risk - that is, the number of extra lung cancers for a given exposure and a given period at risk. For the cohort as a whole, the value was 20 per WLM per 10^6 person years. Different patterns of exposure appeared to affect the risk, although the criteria for assigning men to the

categories were not explicitly stated. For the 70% of men for whom smoking status was obtained, the combined effects of tobacco and radon exposure were reported to be nearly additive. In addition, the authors noted that a significant excess of lung cancer occurred with exposures below 50 WLM, among men over 30 years old at first exposure

A number of reports have been published of uranium miners in the Colorado Plateau in the United States (Wagoner, 1963, 1964, 1965; Lundin, 1969, 1971, 1979; Waxweiler, 1981, 1983; Whittemore, 1983; Hornung, 1985, 1987). The cohort comprised miners examined medically between 1950 and 1960 and who worked underground in uranium mines in the Colorado Plateau for at least one month before January 1, 1964. This gave a total of 3,362 white and 780 non-white miners although the reports are commonly restricted to results on the white miners. Exposure estimates were based on nearly 43,000 radon daughter measurements on 2,500 mines during the period 1951 through 1968. Both interpolation and extrapolation were used to obtain estimates of the exposure levels in WLM for periods when data were not available. Smoking histories were obtained from each subject at some time between 1950 and 1960 and afterwards and were used to estimate total packs of cigarettes smoked. By 1977, 185 white miners had died of lung cancer compared with 38.4 expected. Various modelling approaches have been used on the data. Whittemore and McMillan (1983) conducted a case-control study within the cohort of miners. They found that a multiplicative model for the risk of lung cancer from smoking and exposure provided a better fit to the data than did an additive risk model. This implied that a relative risk model was the appropriate model of choice. Hornung and Meinhardt (1987) followed the cohort through the end of 1982, providing an additional 69 lung cancer deaths (giving a total of 256). They used the Cox Proportional Hazards Model and predicted excess relative risks between 0.9 and 1.4 per hundred WLM, although there was an exposure-rate effect with low exposure rates more harmful per unit of cumulative exposure. It should be noted, though, that some miners had extremely high exposure rates, ranging up to nearly 1000 WLM per month, giving cumulative exposures of over 10,000 WLM. Indeed, the median cumulative exposure was 430 WLM for the cohort.

The final cohort consists of miners in Ontario, who are the focus of this report. Muller et al. (1983, 1987, 1988) constructed a cohort of miners among whom were men with at least half a month of dust exposure in uranium mines in the province and who were employed after 1954 (the mines began operation in 1955). The data used were based on the medical examinations required every year for all miners in Ontario. Altogether, there were 15,984 uranium miners who met the entry criteria. Men were followed through December 31, 1981 (or death). Since an increase in lung cancer in gold miners had been demonstrated, the cohort was divided into those with and without previous gold mining experience. Roughly 1,100 miners were excluded since they worked at some time in a uranium mill, an asbestos mine or in a uranium mine outside Ontario. This left 10,661 uranium miners who had not previously worked in gold mines and 4,216 who had done so.

Since 1955, measurements have been made of radon daughter levels. Extrapolation was used when necessary to estimate radon daughter levels in the early years of mining. Indeed, Muller used two sets of WLM levels - the Standard values, being the best estimate of the actual exposure; and the Special values, showing a reasonable upper limit for the likely exposure. An overall excess of lung cancer was observed, and a modelling approach was used to investigate the relationship with radon exposure. One approach adopted a simple linear function relating excess risk with cumululative WLM (with a five year lag). A second approach employed time since exposure (not the same as time since first exposure). This implies that the risk at any time depends on the pattern of exposure in the past, rather than simply on the total exposure. The authors examined these categories - exposures accumulated five to nine years, 10 to 14 years, and 15 and more years before observation. They found that exposures accumulated 15 and more years ago contributed little to the risk function, whereas the exposure 10 to 14 years ago was the most important and gave a coefficient roughly double that of WLM accumulated five to nine years ago, i.e. such exposures double the effect.

The BEIR IV Committee adopted a similar approach to examining the data from the four cohorts of miners for which suitable data on radon daughter exposures were available. The cohorts were cohorts of miners from Czechoslovakia, Colorado plateau, Saskatchewan and Ontario. The raw data from each was made available for analysis. After investigating a variety of factors, they concluded that the probability of dying of lung cancer at age a in the cohorts was best described by the expression:

$$r(a) = r_0(a) [1 + g(a) (W_1 + 0.5W_2)],$$

where r(a) is the lung cancer mortality rate at age a;

$r_0(a)$ is the "baseline" lung-cancer mortality rate at age a;

g(a) is 0.03 for ages less than 55 years, 0.025 for ages 55-64 years, and 0.01 for ages 64 years or greater;

W_1 is the cumulative radiation exposure in WLM from five to 14 years before age a; and

W_2 is the cumulative exposure in WLM 15 years or more before age a.

This model was based on a pooled analysis following separate examination of the data in each of the four cohorts. It was noted that, for a factor to be incorporated into the model, results had to be consistent across the cohorts.

The BEIR IV Committee considered the combined effects of radon daughters and cigarette smoking (in Appendix VII, pp. 504-563, of its report). The Committee reviewed the epidemiological literature on the health effects from both carcinogens; it performed its own analyses of lung cancer occurrence in persons exposed to both substances; and it

presented its views on the combined associations with lung cancer. The Committee's analysis of cancer risk from radon exposure showed an association with cumulative dose, age and time since exposure. They suggested that other factors influencing the cancer risk might include age at first exposure, dose rate, sex, diet, and genetic predisposition. Association of tobacco consumption with lung cancer depends on duration and number of cigarettes smoked per day, type of tobacco product, method of inhalation, and years since cessation of use for former smokers. Assessment of the combined effects of cigarette smoking and radon progeny is bound, therefore, to be extremely complex.

The Committee's assessment of the current data for U. S. uranium miners suggest that these risks do not combine additively on the relative risk scale; rather, the multiplicative model appears to have greater support in the literature. The analyses by Whittemore (1983) and the BEIR IV Committee of U.S. uranium miners support this latter conclusion.

Muller (1987, 1988) reported the results of a 3% random sample of Ontario uranium miners with no prior gold mining experience including 80 men who died of lung cancer. Smoking information was obtained on 140 controls (62%) and on 73 cases (90%). Each case of lung cancer, for whom a complete smoking history was known, was matched randomly to a control with known smoking history who, at the age when the case died, had the same cumulative exposure in the interval 10 to 14 years ago. The relative risk of smoking was calculated in miners with and without WLM exposure in this interval (10 to 14 years previously) and no significant difference in this risk was found. The authors note that the results are compatible with a multiplicative model for the joint effect of smoking and exposure to radon progeny, although other models might also fit the data. There were too few non-smoking cases to establish a clear preference for any model.

SECTION 2

BACKGROUND INVESTIGATIONS

2.1 STAFF CONTACT WITH CANADIAN AGENCIES

On November 26-27, 1986, Panel staff visited the regional offices of the Atomic Energy Control Board (AECB) in Elliot Lake where records are maintained on radiation exposures for Elliot Lake district uranium miners. An overview of radiation record keeping in Canadian uranium mines was provided by AECB staff. The AECB's methodology for estimating cumulative WLM exposures for uranium miners was described and illustrated with data showing the differences in results obtained between the AECB and the WCB approaches. There was no material difference between the estimates for exposure derived from the two approaches.

In Canada, there were several major areas for uranium mining, notably Elliot Lake in Ontario (including Rio Algom and Denison Mines; and Agnew Lake), the Bancroft area, also in Ontario (including the Greyhawk, Canadian Dyno, Faraday and Madawaska mines), Northern Saskatchewan (including Beaverlodge, Amok, Key Lake and Eldor mines), and the Northwest Territories (Port Radium).

The AECB office in Elliot Lake maintains complete data on individual miners' exposures from 1968 onwards for the Rio Algom and Denison companies. Some additional information is available prior to 1968. The AECB also had complete written employment records for the Madawaska mines (closed in 1983) in the Bancroft area; and some information on Agnew Lake (closed in 1980).

Panel staff also visited AECB headquarters in Ottawa (on December 16, 1986) and met with Al Dory, Manager of the Uranium Mining Division, and his staff. Dory and his staff described the ventilation conditions in mines (or lack thereof) for different categories of mining jobs, time periods, mine locations and ore grades. Working environmental conditions varied tremendously. For example, drillers at the end of a heading could experience marginal advantages due to the air circulation about them provided by the drilling equipment; whereas the slushers immediately behind them would be working in dead air pockets which permitted rapid buildup of radon and radon daughter levels. Similarly, ventilation ducts were usually not extended close enough to the end of headings and therefore there were often dead air pockets beyond the end of the ducts. Additionally, mined-out stopes were a source of radon daughter buildup because of poor air circulation. Consequently, exposure estimates for individuals could never be accurate.

On the other hand, average measurements would tend to be accurate reflections of average mining conditions and exposures for large groups of workers (e.g. for epidemiological studies), but the AECB prefers to

use a high/low range of desired exposures for individual miners between which a given miner's actual exposure would fall. The AECB's average calculations in most cases match closely (in a statistical sense) those for the Standard WLM estimates (in Muller, 1983). The AECB's high estimates are akin to Muller's Special WLM estimates.

It is believed that the Mining Master File (MMF), maintained until recently by the Ontario Workers' Compensation Board, is the most complete record of employment available for Ontario uranium mining. Fifty individual estimates of cumulative exposure were used to compare the methods for deriving individual exposures used by Muller and the AECB. Apart from two zero Muller estimates (from the Stanrock Mill which was ventilated using underground exhaust air laden with radon and radon daughters), there was no disagreement.

Panel staff also met with Dr. Pat Ashmore (on December 16, 1986), Head of the National Dose Registry, Occupational Radiation Hazards Division, Bureau of Radiation and Medicine Devices, Health and Welfare Canada, in Ottawa. The Registry started in 1951 with the mandatory dosimetric recording of various workers (e.g. dentists, X-ray technologists, reactor workers). Dosimetry measurements captured X-ray, gamma and beta ray whole body dose levels. By 1974 or 1975, tritium levels were also being captured for Ontario Hydro workers. Radon daughter exposures for uranium miners were recorded from 1977 on. Prior to 1977, there is only spotty coverage of uranium miners' exposure in the Registry. However, there are lifetime records for Saskatchewan miners. Ashmore plans to merge the MMF data with the Registry in the future.

Registry information is reported quarterly for each exposed worker and shows his WLM for the last calendar quarter. From 1981 on, gamma monitoring began as well for uranium miners. Yearly averages for gamma exposure among uranium miners is about 200-250 millirems. Information is keyed into the Registry by SIN number.

The Wigle group (in the Laboratory Centre for Disease Control of Health and Welfare) is conducting an epidemiologic study of the Registry data base with the initial follow up period extending from 1951-83. The death linkage was to have been conducted in late 1987 with results expected by late 1988 or early 1989. The same group produced a mortality study of nine Northern Ontario communities (showing elevated lung cancer mortality risks for men and women in Timmins); and an update on Newfoundland fluorspar workers.

2.2 MEETINGS WITH MINING ADVISERS:

The Terms of Reference for the Special Panel required advice to be provided by two nominees, one each from employer and employee parties of interest, who would possess significant knowledge and appreciation of the historical conditions in the Ontario uranium mining industry. Accordingly, a meeting was held with Albert Kanabe, Chairman of the Joint Occupational Health and Safety Committee at Elliot Lake, who was

nominated by the United Steelworkers of America; and with Professor Russell Thompkins, Queen's University, who was nominated by the Ontario Mining Association, on November 25-26, 1987 at Panel headquarters.

Prof. Thompkins is a former teacher and consultant who designed the original ventilation scheme used in many uranium mines throughout Canada and elsewhere. Mr. Kanabe has a long working experience in uranium mines from 1957 and is currently Health and Safety Chairman with the United Steelworkers Union in Elliot Lake.

Prof. Thompkins stated that early ventilation practices were relatively unsophisticated as there was no estimation of air requirements for uranium mines. A simple rule of thumb in the early days of uranium mining was to double the amount of air being used in gold mines which was about 50 cfm per ton of ore mined daily. Thus, air flow in uranium mines began initially with about 100 cfm per ton of mined ore and has increased gradually to almost 500 cfm per ore-ton today.

Mining ventilation is achieved using primary and auxiliary circuits for air passage. Primary ventilation involves a continuous path for air flow from (surface) intake to (surface) exhaust; whereas auxiliary ventilation provides for the circulation of air in, for instance, dead end headings using such devices as piping either to push air in or to suck it out and back into a primary ventilation circuit. When headings are connected to other mine areas and air passes freely through them, they become primary circuits. Usually, the ventilation regulations for diesel fumes were sufficiently stringent to ensure that the radon gas regulations were met.

Shallow mining operations, such as the Nordic and Quirke mines, were relatively easy to ventilate. Auxiliary systems in these and other mines would typically move between 3,000 cfm of air (using 24 inch fans) and 50,000 cfm in large drift headings using diesel equipment. Originally, mines in the Elliot Lake areas were ventilated at the rates of from 75,000 to 100,000 cfm. At Quirke and Nordic Mines, their shallow depth enabled exhaust raises to the surface to be quickly and economically established. At other mines, once the two shaft openings were completed and connected, mining took place close to the primary ventilation circuit.

As the mining operations proceeded deeper and further from these simple circuits, production became more complex and larger areas had to be ventilated. Therefore, the volumes of air being used had to be constantly increased to keep the working areas below the 1 WL (working level) target concentration for radon. In 1968, a new initiative to decrease the allowable concentration to 0.33 WL began. Without any other changes, air volumes would have to increase threefold necessitating the sinking of many new air shafts. Enough had been sunk by 1972 to make possible the new regulations in Ontario.

Today, the mines have a complicated system of primary ventilating circuits with several fresh air intakes and exhaust openings. The main principle underlying these systems is that fresh air is delivered to the working stope areas of the mine so that no more than three stopes in succession are ventilated before the air is directed to a principal exhaust circuit. The mined out portions of a mine generally serve as a vast collection area for vitiated air leading to the several exhaust raises. At Denison, for example, 2,000,000 cfm is exhausted from one opening through a large vertical fan. In winter, Denison Mine heats a total of 5,000,000 cfm supplied to the mine because no air may be recycled within the mine. In addition, all bulkheads used to control the air circuits within the mine are kept under pressure so that leakage is drawn to the exhaust circuits.

Stanrock Mines was a notable exception to the system described for Elliot Lake because fans were located in the middle of its ventilation cicuits leading to considerable leakage and recycling of air in that mine. Subsequent exposure records revealed consistently higher WL readings (in later years) than the other mines. The Bancroft area mines also averaged higher radiation levels than the Elliot Lake mines, particularly Denison and Rio Algom.

Some concern had been raised about whether mills were heated with air from the mines. Prof. Thompkins commented that only Stanrock Mines followed this practice. This mine has been closed since the late sixties. No mine recycles air on a regular basis.

Air sampling was designed to find areas where the allowable concentrations were being exceeded so that corrective action could be taken quickly. This grab sampling technique required that every working place be sampled at the highest possible frequency. Although grab sampling was not the best method to establish exposure levels, it was the only one available. Because frequent samples were taken, Ontario probably has the best historical records of radon exposure for uranium mining available anywhere in the world.

Radon is perceived to be the major hazard in uranium mines. Most radiation (about 90%) comes from broken ore rather than being emitted from the walls. Such ore is therefore removed from the mine as soon as possible since concentrations continue to increase for about 28 days in the pores of an unremoved ore pile.

Because the ore body in the Bancroft area uranium mines was more vertically oriented, a shrinkage stope mining method was generally used which always resulted in the retention of some broken ore in the stopes. For this reason, it was more difficult to keep radon concentrations low than at Elliot Lake where ore was removed as rapidly as it was broken.

In the 1950s, there were diverse ethnic groups among the miners including Yugoslavian, German, Finnish, Scandinavian and British

immigrants. Movement of workers among different mines was very common.

Exposure to radon or other contaminants varied with the job performed. In slushing, for instance, the best air was at the bottom of the tunnel where work was being done. Prof. Thompkins commented that this type of ventilation system was based on the 'last man exposure design' whereby the last man in a particular process received no more than the allowable amount of gas and dust while others in the same process received less. Leaching, when first started, involved the conventional mining of worked-out stopes. The exhaust air passed through these stope areas which always contained higher than normal WL. However, workers serviced these areas infrequently and were protected by face masks.

Besides radon and radon daughters, other exposures in mines included gamma rays, diesel fumes and oil associated with drilling. Oil (RD 125) was the lubricant used in air drills. During drilling there was often a fine oil mist which, combined with dust, could be inhaled. Silica dust levels in the air were greatly affected by blasting. General blasting was always done at the end of a shift, a practice which began in gold mining in the early 1940s. It could only be carried out with the special permission of the mine captain so as to minimize the effect on other workers.

All mines implemented aluminum prophylaxis treatment. Ten grams of aluminum powder for every 10,000 square feet of air were pumped into the change room during the period when miners changed clothing. It was believed that miners' inhalation of the aluminum would inhibit development of silicosis. The absence of follow-up studies to prove its effectiveness led to its abandonment in 1979 as a result of workers' demands.

An attempt was made by both advisers to account for differences in sample readings. Whereas neither adviser questioned the integrity of the sampling procedures, both agreed that variances in readings might be accounted for by:

- grab vs. continuous sampling;
- differences in sampling technology and accuracy;
- different people taking the sample in physically different locations.

Since 1981, both company and union representatives have been taking similar samples and investigating any discrepancies in readings. Both advisers lauded this practice.

As noted above, the maximum permissable radon level was reduced in 1972 from 1.0 to 0.33 WL which required significant changes in ventilation practices. Mr. Kanabe believed that, in general, industrial hygiene has improved since 1972. Since the reduction (to 0.33WL) had occurred first in the United States in 1967, Canadian uranium mining companies had time to install additional ventilation schemes to meet the reduced

exposure limit by 1972. Changes in blasting techniques have also improved hygiene levels. The replacement of dynamite with ammonium nitrate in the mid-1970s made possible 'central blasting', in which all miners leave the mine before blasting. None returned to any work area immediately after a blast. Secondary blasting of large pieces of muck to reduce the pieces to a manipulable size was carried out at the end of a shift to reduce radon exposure. An increase in radon concentration would only occur during a primary blast to produce new broken ore. It would occur for only a few minutes and it would be quickly removed by the ventilating system before a new shift began.

Mr. Kanabe noted that, until 1979, the problem of diesel fumes was often severe at mine headings. Prof. Thompkins noted that this could be accounted for not only by the presence of too many machines in the area, but also by ventilation design problems. As well as carbon monoxide, diesel fumes also contain aldehydes. In general, the problem of diesel exhaust was a function of the type and size of machines as well as their age and general condition of maintenance.

The advisers tabled various documents during the discussions and these are listed in Appendix B, the Evidentiary Base. Among those tabled by Prof. Thompkins were: a comprehensive three-part article on radiation in uranium mines (Thompkins, 1982) and risk estimation (Anon; Stewart, Aug. 27, 1976; Stewart, July 28, 1976); and articles on mine design (Thompkins, math. sim.), mine ventilation routes (Thompkins, maps) and silica dust levels (Rio Algom, 1984). Among the documents tabled by Mr. Kanabe were items on: the toxicity and extent of diesel emissions (Bardswitch, 1979); a materials safety data sheet for Ardee 100, a lubricant and coolant for rock drills (Gulf Petro Canada, 1987); and items concerning radiation sampling (Ont. Min. of Labour, 1977; Ont. Min. of Nat. Res., 1976; Ont. Dept. of Health, 1958); and air quality (Rio Algom, 1976; Rio Algom, various dates).

SECTION 3

PREVIOUS GOLD MINING EXPERIENCE AMONG URANIUM MINERS

3.1 INTRODUCTION

As noted earlier, one focus of the present report is to take into account prior gold mining experience among uranium miners. Gold miners have been found to have an increased rate of lung cancer (in the absence of uranium mining) and a previous report of the Panel (IDSP, 1987) found a dose-response relationship between a "weighted" dose of dust in gold mines and excess risk for lung cancer. The weighting was designed to take account of varying dust conditions over time in the gold mines. Thus, duration of dust exposure before 1936 was assigned a weight of four; between 1936 and 1944 a weight of three; between 1945 and 1954 a weight of two; and from 1955 onwards a weight of one. To some extent, the relationship between this dust index and lung cancer risk also reflects a relationship between time since first exposure (latency) and risk, but the two variables (dust index and time since first exposure) are highly correlated, so that they are readily interchangeable.

The latest report by Muller and Kusiak (1987, 1988) distinguishes uranium miners with and without prior gold mining experience. However, the distinction was simply dichotomous and took no account of the extent of exposure to dust in gold mining. It was decided for this report that such allowance was necessary, so a modelling approach was used to derive an estimate of "background risk" for uranium miners with prior gold mining experience. The technique used is known as Poisson regression (described below), and yields estimates of the Standardized Mortality Ratio (SMR) or relative risk for men with a given gold mining profile relative to the provincial population. (The SMR is the ratio of the obseved number of deaths to the number expected if the mortality experience in the cohort were the same as that for the Ontario male population.)

3.2 ON THE POISSON REGRESSION TECHNIQUE

Models describing lung cancer risk for miners can be derived using statistical analyses which incorporate various characteristics such as the dust index and time since first exposure for gold miners; and age at risk and previous exposure to radon daughters for uranium miners.

The statistical analyses are based on the following propositions. It is assumed that the number of deaths of cohort workers over a period of time can be characterized as a random variable with a Poisson distribution. The Poisson distribution is used to describe the probability of observing a given number of deaths in a sample drawn from a population in which the "average" number of deaths is known. The Standardized Mortality Ratio (SMR) provides a comparison for a given cause of death of the observed and expected number (and rate) of

deaths from that cause. As noted earlier, the expected number is calculated assuming the mortality experience (or rate) of the study group is identical to that for the population from whom the group is drawn (e.g. the general population of Ontario).

In a cohort study, each qualifying member is followed forward in time until the closing date of the study or until a defined health event occurs (e.g. mortality), whichever comes first. In this manner, the member accumulates a certain number of person years (PYRs) during each of which he is "at risk" of the given health condition. Each PYR is defined by: a given calendar year, age at risk, sex, geographical location and so on. Moreover, for uranium miners, each PYR is further characterised by the: cumulative radiation exposure to date (in WLM), radiation exposure in a period of time 5-9 years previously (or 10-14 years, or 15+ years ago). For uranium miners with previous gold mining experience, each PYR is associated with, in addition, the following measures: time (in years) since first gold dust exposure, calendar year of first gold dust exposure and cumulative weighted gold dust exposure (previously defined).

Each PYR is associated with a risk of dying from lung cancer (for a given age at risk, sex, calendar date and geographical location - in this case, Ontario). For each PYR, the (fractional) number of expected deaths can be calculated by applying the corresponding cause-specific mortality rate for Ontario (age-, sex-, and calendar date specific rate). In this manner, the mortality experience for the cohort can be grouped in various ways in order to examine the relationship of the different descriptive factors on the risk of lung cancer. One particular grouping would be by cumulative WLM exposure. For each interval in the range of possible exposures, there is an observed and expected number of lung cancer deaths. We assume that the data in each cell is Poisson distributed as are the total deaths from lung cancer for the entire cohort. The expected numbers are based on rates from a large population.

The relationship between relative risk for lung cancer (as measured by the SMR) and any descriptive variable can also be modelled using regression techniques. Because epidemiological cohort data can be considered to be Poisson distributed, recourse was made to a statistical program called the Generalized Linear Interactive Modelling System or GLIM for short (The GLIM System, Release 3.77. Royal Statistical Society, 1987). This program enables the estimation of linear relationships between observed risk estimates and descriptive variables on the assumption that the underlying error distributions are Poisson in nature.

Among the statistics generated by GLIM in any given estimation procedure is the deviance. This quantity can be interpreted as the residual sum of squares in an ordinary multiple regression in which the underlying distribution is normal. Under certain regularity conditions, it has been shown that the deviance is distributed as a chi-square with an appropriate number of degrees of freedom. Usually,

one measures the reduction in deviance resulting from the introduction of additional descriptor variables in which case the degrees of freedom in question equal the number of additional parameters being estimated. (The interpretation of the deviance where normality does not apply is as chi square only asymptotically. It is known, however, that the Poisson distribution can be approximated by a normal distribution for expected values greater than 5 or 6. Grouping procedures that result in cells with expected values of this size or greater would allow for the interpretation in an asymptotic sense of the distribution of the deviance as chi square.) The models of lung cancer risk described below and in the associated tables have been estimated using the GLIM statistical package.

3.2 GOLD MINERS' DATA REVISITED

Data from the gold miners' study (IDSP, 1987) were reanalyzed to obtain models relating lung cancer risk with various previously identified risk factors. These gold miners qualified for inclusion in the cohort if they were working currently in a gold mine at the time of their miners' chest X-ray exam between January 1, 1955 and December 31, 1976; and had at least 5 years of gold dust exposure including 0.5 months of Ontario gold mining. Person years for this group were excluded after a man started work in a uranium mine.

Factors considered as possible modifiers of risk were:

- weighted gold dust exposure;
- time since first exposure;
- calendar year of first exposure; and
- age and period at risk.

The latter two factors (age and period at risk) were found not to contribute descriptive power to the model of lung cancer risk. Each of the other three was significant, not surprisingly, since they are highly correlated.

The results of the modelling, expressed as equations showing SMRs as a function of predictor variables, are given in Table 1. The predictor variables include cumulative weighted gold dust exposure, calendar year of first exposure and time since first exposure. A number of observations concerning these risk models for lung cancer among gold miners in Ontario are worth making. The evidence points to dust conditions in the mines as the occupational factor most likely responsible for the disease. These conditions appear to have largely abated since 1945. Models (1) and (5) (with the dust index) suggest an 0.8% increase in relative risk for every unit increase in the index. Model (2) (with calendar year of first exposure) suggests that occupational risk had essentially disappeared by about 1949. Of course, these results reflect the information in this cohort dataset. With a new update extending the follow-up period of these miners from 1976 to 1985 (expected in the near future), a more complete picture of the risk of lung cancer among Ontario gold miners should emerge.

SECTION 4

DEFINITION OF THE URANIUM MINING COHORTS

The cohort was drawn from the Mining Master File (MMF), maintained until recently by the Ontario Workers' Compensation Board. The MMF contains detailed employment histories for all Ontario miners as provided by those miners during their required annual chest examinations. The minimum exposure required to qualify for uranium mining was half a month. Thus the exact criterion for admission to this study was as follows:

Attendance at a miner's chest X-ray examination at any time between January 1, 1955 and December 31, 1981 at which time the miner reported at least one half month's employment in an Ontario uranium dusty job (defined by the WCB job codes: 11-16, 21, 22, 25, 26 and 97) and current employment in an Ontario uranium mine. Person-years were counted from the date of admission to the study.

Miners were excluded from the study if they had prior asbestos or extra-Ontario uranium mining or milling experience. After entering the study, a miner (and his associated person years) was removed from the study after the point at which he entered known asbestos exposure or uranium exposure outside Ontario. Thus, a miner who began a dusty uranium job after asbestos work was excluded altogether while, for a man who had asbestos exposure after uranium mining, person-years were counted only up to the date of first asbestos exposure. Otherwise, follow-up continued until the end of 1981, or until death if that occurred earlier.

This definition differs from that used by Muller et al. in two respects. Firstly, the latter did not require that a man be in uranium mining at the time of his examination. However, since miners who left uranium mining for other parts of the mining industry (but who nevertheless reported their uranium mining experience at their annual exam) might be different from those who left the mining industry altogether after their uranium mining (and therefore would not have reported their uranium mining experience), the requirement of current employment in uranium mining was preferred. Differences resulting from this change would be expected to apply only to workers with short term employment.

Secondly, this study was based solely on the miners' reports at the time of their examinations. Muller et al. included men identified as underground uranium miners using only their exposure records as reported to Atomic Energy Control Board authorities. These reports were a regulatory requirement from 1968 onwards.

A total of 14,373 uranium miners with 221,806 person-years at risk qualified for the study and comprised the "ALL" cohort (Table 2). A number of subdivisions of this cohort was made. The "NOMILL" cohort (No. 2) counted person-years at risk for each uranium miner (ie. all

those in the "ALL" cohort) until they began work in a uranium mill (WCB job codes 11 & 21). This definition thus excluded those who worked in a uranium mill before a dusty job elsewhere in uranium mining. The "MILL" cohort (No. 8) counted person years after entry into milling. Lack of information on radon exposure in uranium mills made this distinction necessary.

Further subdivisions of the cohorts were based on a miner's exposure to other ores. This was necessary for two reasons. Firstly, because gold miners have now been shown to be at increased risk for lung cancer, account of employment in this industry was made. Secondly, a previous report (IDSP, 1987) had shown a slight difference in lung cancer risk for men exposed to "other" ores, which made necessary a similar check for the uranium miners as well.

The GOLD cohort (No. 3) included all person-years for uranium miners without mill exposure (the NOMILL cohort, No. 2) after their first gold-dust exposure. In contrast, the NOGOLD cohort (No. 4) counted all person-years for the NOMILL miners before their first exposure to a dusty gold mining job. Of course, if a uranium miner never received any gold mining exposure, his person years were allocated only to the NOGOLD cohort until he left the study. The GOLD cohort was further subdivided into two groups, the GOLDBE cohort (No. 3A) had their first dusty gold-mining exposure either before or in the same year as their first dusty job in uranium mining; while the GOLDAF cohort (No. 3B) comprised men whose first dusty exposure in gold mining followed their entry into the uranium cohort.

The remaining sub-cohorts were designed to examine the effect of exposure to other ores, by removing from the NOGOLD cohort those person years following exposures to other ores in the mining industry. Thus, the NONICU cohort (No. 5) consisted of person-years for men in the NOGOLD cohort until their first exposure to nickel/copper ore after which all remaining person-years were omitted. For those men who never worked in nickel/copper mining, person years would be counted in the NONICU cohort until death or the end of 1981. Cohorts No. 6 (NOIRON) and No. 7 (NOANY) were defined similarly. Table 2 shows the number of miners and person-years in each cohort.

The GOLDAF cohort (No. 3B) was small and, although there was a small but statistically significant excess number of lung cancer deaths in this cohort, it was assumed that radon exposure rather than the effect of gold mining after uranium mining was the underlying cause. Gold mining for this cohort would always have been after 1954 when a previous study (IDSP, 1987) had not demonstrated any excess lung cancer risk among gold miners. Therefore, further analyses combined person-years in this cohort with those in the NOGOLD cohort (No. 4) to form the NOGOLDBE cohort. These person-years were non-overlapping with the GOLDBE COHORT (No. 3A).

These definitions make it clear that an individual miner could contribute person-years to different cohorts depending on his work experience. Miners who left Ontario to work in gold or uranium mines elsewhere and did not return to the province would not have had that exposure recorded in the Mining Master File. To the extent that this happened, the exposures recorded for men in the cohort underestimate their total exposure.

SECTION 5

COHORT ANALYSES FOR URANIUM MINERS

5.1 PRELIMINARY REVIEW

The first analysis involved the validation of deaths certified as lung cancers in the NOMILL cohort (some 156 cases) through a comparison of their death certificate diagnosis with corroborating information in the Ontario Cancer Registry (Table 3). Only cases for which the Registry had additional information, such as pathology reports, were considered. Additional data was obtained for one hundred two (102) of the 156 cases. The extra information confirmed the death certificate cause of death in 99 cases for a validation rate of 97.1%. (The data in the Registry are computerized from 1964 onwards, so that some earlier cases could not be reviewed). When a similar comparison was made for the Registry as a whole, the validation rate was 93%. Thus, as Table 3 suggests, there is, if anything, a slight underestimate of mortality risk for lung cancer (about 4%).

The next analysis consisted of calculating mortality tables for the nine cohorts by cause of death (Tables 4 through 13). These tables display observed and expected deaths by cause, the associated Standardized Mortality Ratios (SMRs) and their two-tailed p-values (SMRs were age-, sex-, and calendar period standardized). Overall mortality is about 10% greater than expected (in the ALL cohort), a significant increase. This increase is more than accounted for by an excess of deaths due to "accidents, poisoning and violence". Among disease causes of death, there is a deficit of deaths for diseases of the circulatory system. Among malignant neoplasms, there is a significant excess of deaths due to cancer of the trachea, bronchus and lung. There is also an increase in pneumoconioses (under the category labelled "chronic interstitial pneumonia"). In addition, there is also a slight, but non-significant, increase in stomach cancers (see especially Table 7 for the GOLDBE cohort), as was found earlier for gold miners without uranium experience. Further, and importantly, there is no suggestion of an increase in lymphatic and hematopoietic tissue cancers, a category that includes leukaemia; nor is there any significant excess of kidney cancer.

An examination of the lung cancer mortality excess in each of the separate sub-cohorts shows it to be significant in all but the MILL cohort, although this group was very small. Two studies by Muller et al., (1983; 1987) showed an excess of lung cancer deaths in their EVERMILL cohort. The difference is probably due to the difference in cohort definitions.

The SMR for lung cancer was lower in the NOANY cohort of men with no recorded exposure to "other" types of ore (Table 12). To investigate this observation further, the NOGOLD cohort was subdivided into "ANY" and "NOANY" sub-cohorts. A NOGOLD miner remained in the NOANY sub-cohort until he entered any "other ore" mining after which his person

years were counted in the ANY sub-cohort. Table 14 shows the lung cancer mortality risk by cumulative WLM exposure for the ANY, NOANY and NOGOLD cohorts. Nearly all of the excess lung cancer deaths for the NOGOLD miners are clustered in the ANY cohort. It confirms that uranium miners had some of their uranium mining experience incorrectly labelled as any "other ore" experience.

Subsequent stages in the analysis focused on the GOLDBE and NOGOLDBE cohorts, previously defined. Table 15 shows the distribution of mortality risk from lung cancer by age at death for these two cohorts. There is a progressive increase in age-related risk for the GOLDBE miners. However, for uranium miners with no previous gold mining experience, the mortality risk from lung cancer is concentrated in the two age groups under 65. The model derived by the BEIR IV committee (referred to in Section 1.2.4 above) also suggested that risk was related to age. The relationship, though, differed from that reported here. Their model showed lung cancer risk increasing from the <55 years age group to the 55-64 age group, and then dropping for the 65+ age group. Our NOGOLDBE data show no increased risk for miners over the age of 64. One possible explanation is that the Ontario uranium miners are a young cohort with an under-representation of men in the older age groups. For this reason, the cohort at this time has little power to reveal any significant excess of lung cancers among older miners, and the estimate of effect in this group has a large standard error. Future follow-ups extending the period of observation of these miners will resolve this issue.

Tables 16 through 20 display the gold mining characteristics of the GOLDBE cohort. The factors shown (including duration of gold dust exposure in total and prior to 1945, calendar year of first gold dust exposure, weighted gold dust exposure, and time since first gold dust exposure) were examined previously for gold miners without uranium dust experience (IDSP, 1987). Tables 16 and 17 show increasing lung cancer mortality risk among uranium miners with increasing duration of gold dust exposure both before 1945 and overall. However, the risk gradient is less pronounced for overall duration (Table 16) as was the case for the gold miners' cohort. Table 18 (for calendar year of first gold dust exposure) shows a pronounced lung cancer risk gradient with declining risk the later the first exposure year. The stomach cancer pattern is the reverse and so appears biologically inconsistent with an occupational association. These results are again in accord with previous findings for gold miners. Table 19 (for weighted gold dust exposure) and Table 20 (for time since first gold dust exposure) show an increasing lung cancer risk gradient with increases in either predictor variable. The stomach cancer pattern for these variables is, however, contradictory. These latter results again duplicate the earlier gold miners' results. In summary, the same general pattern of lung cancer risk found earlier among gold miners appears to also apply to uranium miners with previous gold dust exposures.

Lung cancer risk according to cumulative radiation exposure was further examined for each of the GOLDBE and NOGOLDBE cohorts (Table

21). In both, there is an increase in risk with increasing cumulative exposure.

This was tested formally using the method of Poisson regression (described in Section 3.2 above). It was assumed that the risk would increase linearly with exposure, that is, that for any given increase in cumulative exposure, the relative (or attributable) risk would increase by a fixed amount. For each of the NOGOLDBE amd GOLDBE cohorts, the first five years of follow-up were excluded. The cohort data were then organized by exposure category as shown in Tables 22 and 23. Mean exposures (within each interval) were calculated using PYRs as weights. The relative risk for each exposure interval was calculated as the ratio of observed to expected deaths. The attributable risk was also calculated as the ratio of excess deaths to PYRs in each category all multiplied by a scaling factor (10^6). The regression for RR was estimated using an intercept of unity for zero exposure; while the AR regression used a zero value for zero exposure. All regressions were significant. The relative risk coefficients for each of the NOGOLDBE and GOLDBE cohorts were 1.76% and 1.63% per WLM respectively. The attributable risk coefficients were 6.58 and 11.36 per WLM per 10^6 person years. These coefficient estimates lie between corresponding values in Howe (1986, 1987). Howe's relative risk coefficients were 0.27% and 3.28% per WLM for his Port Radium and Beaverlodge cohorts respectively; while his corresponding attributable risk coefficients were 3.10 and 20.80 respectively. The average exposures at Port Radium are known to be much higher than those for the Beaverlodge mine which latter are more comparable to Ontario uranium mining exposures. The indicated estimates for the GOLDBE and NOGOLDBE cohorts appear, therefore, to be consistent with other reported Canadian studies.

5.2 DETAILED ANALYSIS OF LUNG CANCER RISK IN URANIUM MINERS

5.2.0 Modelling Approaches

The literature review (Section 1.2) contains an expression for lung cancer risk derived by the BEIR IV committee. The expression relates the risk at a given age to a product of the background risk (the population lung cancer rate at that age) and a function of the age at risk and exposure to radon daughters in different periods before the period at risk. Muller et al. (1987) reported a similar expression for the uranium miners with no gold exposure. However, for those miners who had previously worked in gold mines and for whom there was excess risk of lung cancer, some adjustment for gold dust exposure was required in the modelling treatment.

Two approaches were considered. Firstly, the results of the previous gold mining analysis (Table 1) for miners with no uranium mining experience were used to estimate a model of lung cancer risk for uranium miners with previous gold dust exposure. Previously defined risk factors for gold miners were incorporated into an adjustment of

the background risk before considering the additional effect of uranium mining.

A second approach was to construct a model that incorporated simultaneously the effects of both gold and uranium mining. In this analysis, all the data for both the GOLDBE and NOGOLDBE cohorts were combined. This was in contrast to the first method for which the two sub-cohorts were treated separately.

Both methods were adopted. In the first, the risk of lung cancer following gold mining exposure was modelled using several variables as potential predictors of risk: age at risk, period at risk, year of first exposure, time since first exposure, and weighted dust exposure. The modelling was accomplished by using the observed and expected numbers subdivided into risk categories according to the predictor variables shown in Table 1. These variables are not all necessarily causal factors for lung cancer risk; but rather they provide descriptive power concerning the variability in the risk. Thus, age per se is not a causal factor although it is true that the background risk of lung cancer increases with age. The gold mining risk models of Table 1 were then applied to adjust the background risk for the risk of lung cancer from gold dust exposure. Subsequently, the additional effect of uranium mining risk was estimated using each of two factors:

Factor A: lagged exposure variables of WLM exposure 5-9, 10-14, and 15+ years before observation or death;

Factor B: a "BEIR IV Factor" with a categorical variable for age (less than 55, 55-64, 65+ years of age at risk) multiplied by a combined exposure variable expressed as a linear combination of the lagged exposure variables as estimated using Factor A.

The second approach incorporated the risk from gold dust exposure by modelling it directly in combination with the uranium mining experience. The following modelling form was used:

$$r(a,w,y) = r_0(a)\, F(a,w,y)$$

where: $r_0(a)$ is the age-specific background lung cancer rate;

$r(a,w,y)$ is the age-specific lung cancer rate from radon exposure w and gold dust exposure y; and

$$F(a,w,y) = 1 + [b(a)\cdot w] + [g(a)\cdot y] + [L\cdot b(a)\cdot g(a)\cdot w\cdot y]$$

In the function F, b(a) and g(a) represent (possibly) age dependent radon and gold mining effects. L is a parameter multiplying a combined effect, which allows the model to be either multiplicative (L = 1) or additive (L = 0). Intermediate values of L imply an intermediate effects model, i.e. between additive and multiplicative.

5.2.1 Modelling Prior Gold Mining Exposure: Approach One

The first approach generated six separate models (Nos. 2.1 through 4.2 in Table 24). Another pair of models (Nos. 1.1 and 1.2 in Table 24) are provided for comparison purposes to show the results of this modelling approach when no adjustments are made for previous gold mining exposure. The "BEIR IV factor" (Factor B) differs slightly from its original. In the original model, the factor included two lagged exposure variables (5-14 years and 15+ years prior to observation). The lagged exposure variable 5-9 years ago was found to have no explanatory power. This was also found by Muller (1987, 1988).

The age-related coefficients in the BEIR IV model had the values (0.03, 0.025, 0.01) for the three age groups (<55, 55-64, 65+) respectively. This study yields values varying from (0.007, 0.017, 0.037) to (0.01, 0.015, 0.03), suggesting increasing risk in older age groups. Similarly, the BEIR IV Committee used 0.5 as the coefficient for their lagged exposure variable, implying that the effect of exposure accumulated 15 or more years before the period at risk was 0.5 times that of exposure 5-14 years before. Corresponding estimates in this study varied from 0.2 to 0.5. Note that the coefficient for W_3 in equation (2.2) is 0.432, which value is derived from the ratio of the coefficients for W_3 and W_2 respectively in equation (2.1) (i.e. 0.432 = 0.00785/0.0182).

Because of insufficient representation for the oldest age group (65+ years) in the NOGOLDBE cohort, it was not possible to estimate the exact form of the BEIR IV factor. Two age groups (<55, 55+) and two lagged exposure variables (5-14 years, 15+ years prior to observation) were used. However, the results come surprisingly close to the BEIR IV model (Table 25). The age-related coefficients are (0.023, 0.014) respectively and the corresponding coefficient for the lagged exposure variable was 0.46. These results replicate the model results for the BEIR IV model as well as the data will allow, given that the oldest age group (65+ years) is under-represented.

Lastly, an approach for selecting the "best" model of the lung cancer mortality risk among the GOLDBE miners was considered. Table 26 displays the deviance after the application of the various models shown in Table 24. Using this approach, the best model would be that with the cumulative weighted gold dust exposure in conjunction with the "BEIR IV factor". However, there was relatively little to choose between the different possible models.

To examine this further, the SMR was estimated for each lung cancer case in the GOLDBE cohort with non-zero radiation exposure (86 cases) using as the estimates the models for workers with the same exposure characteristics as the case at his death (i.e. Models 2.1 through 4.2 in Table 24). Thus, estimates of individual risk were obtained for six models (Nos. 2.1 through 4.2). For each pair of models the Pearson correlation coefficient for the predicted SMRs for the lung cancer cases was computed (Table 27). Nine of the fourteen correlations were

0.92 or greater. The lowest correlation was 0.79. All the models rank the lung cancer cases' SMRs (for occupational risk) in roughly the same order.

One additional regression analysis was performed. The models of Table 24 were re-estimated using an intercept of unity for zero exposure (or its surrogate). The results are shown in Table 28. Coefficient estimates for similar models in Tables 24 and 28 are stable which fact suggests that the forms of the models provide useful descriptions of the relationship between lung cancer risk and gold and uranium mining experiences.

5.2.2 Modelling Prior Gold Mining Exposure: Approach Two

Table 29 displays the results of the modelling approach in which gold mining exposure variables and uranium mining factors are introduced additively. No models with a combined interaction term are shown as attempts to introduce this additional term revealed that it provided no additional explanatory power. The greatest reduction in deviance results from the introduction of a cumulative weighted gold dust index (DST) and a "BEIR-IV" factor for uranium exposure (Model 8).

SECTION 6

DISCUSSION AND CONCLUSIONS

It is important to consider possible inaccuracies in the results. These can arise in a variety of ways, notably in measuring exposures and secondly in ascertaining mortality.

A previous report of the Panel used a weighted dust index to measure the cumulative exposure in gold mines. This was an approximation, but fitted the data well. However, as with the radon measurements, there were several reasons why the estimates might have been inaccurate. Firstly, the work histories provided by miners at their annual chest x-ray examinations might have been mistaken. Secondly, the values ascribed were based on average levels for each mine, rather than on the particular job or occupation carried out by the individual. To the extent that such errors are random, the effect on the exposure-response relationship is likely to have been to underestimate the slope of any effect of exposure. It should also be pointed out that, as reported by the Panel's advisers, miners until 1978 worked at a variety of jobs within each mine. Thus, the average exposure level for the mine was probably a good estimate of the typical exposure incurred by each individual.

Calculations of radon exposure in this report are based on "standard" WLM values as reported by Muller et al. (1983). These were believed to be the best estimates of exposures in the uranium mines. The "special" values that Muller reported were not used. These were considered to be upper estimates of the likely exposure in the mines, and were approximately double those of the "standard" values. Thus, had these values been used, any prediction of effects due to radon exposures would be roughly halved.

Errors in the ascertainment of mortality, as well as in diagnosis of cause of death, could affect the results. Muller et al. (1983) estimated that roughly 6% of deaths were not found through the Record Linkage procedure. In addition, the validation exercise carried out at the Ontario Cancer Registry suggest that lung cancer risk estimates based on the Ontario population may have been approximately 4% too high, in relation to the observed deaths among uranium miners. Together, these observations suggest that lung cancer mortality risk may have been understated by roughly 10%.

One might also want to consider whether the increase in lung cancer in uranium mines was due to radon or some other factor. The pattern of risk seems so unique and yet is consistent with results from other studies in which radon exposure was a primary factor, that it is hard to attribute the increase to any other occupational factor. Smoking habits of the cohort were not known, although Muller et al. (1986) found in a case control study of lung cancer cases that a multiplicative interaction between smoking and radon exposure appeared reasonable. It is worth noting that other diseases known to be related

to tobacco use - non-malignant respiratory disease and circulatory diseases - were not increased in this cohort.

The primary aim of this study is now addressed. The evidence confirms that lung cancer risk is increased because of uranium mining exposure, but that no other disease (apart from silicosis) is occupationally related. Cases of silicosis can be regarded as occupationally caused, and accidents on the job are clearly in the same category. However, lung cancer causation is multi-factorial in nature. As a consequence the expressions derived in the previous section for the relationship between lung cancer and uranium and gold mining exposure can be used to rank cases of lung cancer according to the probability that they were occupationally caused. Four equations are considered relevant:

a) For uranium miners with no prior gold mining experience, **equation 1 in Table 25**;

b) For uranium miners with prior gold mining experience, **equation 2.2 in Table 24**, or **equation 2.2 in Table 28**;

c) For the combined group of uranium miners, **equation 8 in Table 29**.

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TABLE 1

GOLD MINERS' COHORT: MODELS OF STANDARDIZED MORTALITY RATIOS FOR LUNG CANCER

MODEL	REDUCTION IN DEVIANCE	REDUCTION IN DEGREES OF FREEDOM
(1) SMR = 1.033 + 0.00786 DST	4.24	1
(2) SMR = 2.588 - 0.0324 YFEP	9.32	1
(3) SMR = 0.389 + 0.03125 TFE	9.00	1
(4) SMR = 1.677 - 0.0197 YFEP + 0.0137 TFE	9.61	2
(5) SMR = 1 + 0.00847 DST		

Notes:

1. Models are estimated using Poisson regression techniques.

2. Notation: SMR Standardized Mortality Ratio;
   DST Cumulative weighted gold dust exposure in dust-years
   where: weight = 4 for years before 1936;
   3 1936-44;
   2 1945-54;
   1 1955+.
   YFEP Calendar year of first exposure - 1900;
   TFE Time since first exposure (in years).

3. Reductions in deviance and in degrees of freedom for the model shown are measured from the model fitted with the intercept alone.

4. Equation (5) was estimated using (SMR-1) as the dependent variable. Thus, no intercept model against which reductions in deviance could be measured was produced in this case.

TABLE 2

URANIUM MINING COHORT CHARACTERISTICS

COHORT		NOS. OF MINERS	PERSON YEARS AT RISK
1.	ALL	14,373	221,806
2.	NOMILL	14,261	220,021
3.	GOLD	5,046	91,365
3A.	GOLDBE	4,664	86,227
3B.	GOLDAF	382	5,138
4.	NOGOLD	9,524	128,656
5.	NONICU	7,516	100,978
6.	NOIRON	9,175	123,137
7.	NOANY	6,542	78,376
8.	MILL	166	1,785

TABLE 3

VALIDATION OF LUNG CANCER CASES USING ONTARIO CANCER REGISTRY

COHORT		VALIDATED	OTHER DIAGNOSIS	TOTAL
GOLDBE		57	2	59
NOGOLDBE		42	1	43
Total:	N	99	3	102
	%	97.1%	2.9%	100%
REGISTRY:	N	29,473	2,189	31,662
	%	93.1%	6.9%	100%

Notes:

1. Only cases appearing in the Registry with death certificates showing lung cancer as the primary cause of death and for which there was additional confirmatory (or otherwise) data in the Registry are included above. Thus, only 102 out of a possible 156 cases (or 65.4%) from the NOMILL cohort had additional data in the Registry. Remaining cases either died out of province or did not have additional data reported to the Registry. Registry data is based on the time period 1964-81.

2. Because the error rate is greater for the Registry data (6.9% versus 2.9% for the NOMILL cohort), there is the possibility that the reported mortality risks are underestimated slightly.

TABLE 4

MORTALITY DATA
COHORT 1: ALL URANIUM MINERS (ALL)

CAUSE OF DEATH	OBSERVED	EXPECTED	SMR	P-VALUE
ALL CAUSES	1352	1218.13	110	.000
ALL DISEASE CAUSES	937	1013.83	92	
1. INFECTIVE DISEASES	9	7.33	122	
Silicotuberculosis	0	0.08	-	
Pulmonary Tuberculosis	3	1.83	164	
Other Tuberculosis	2	0.78	256	.089
All Other Infective Diseases	4	4.65	86	
2. MALIGNANT NEOPLASMS	317	264.60	120	.001
DIGESTIVE ORGANS	77	80.83	95	
Stomach	21	17.94	117	
Intestine or Rectum	35	33.91	103	
Other Digestive	21	28.97	72	
RESPIRATORY SYSTEM	158	89.41	177	.000
Nose, Nasal Cavities, etc.	0	0.83	-	
Larynx	2	4.14	48	
Trachea, Bronchus, Lung	156	83.69	186	.000
Other	0	0.75	-	
LYMPHATIC AND HEMATOPOIETIC TISSUE	21	28.94	73	
OTHER SPECIFIC MALIGNANT NEOPLASMS	61	65.42	93	
Bone	1	1.30	77	
Kidney	5	6.91	72	
Bladder	1	6.09	16	
Brain	10	13.34	75	
Prostate	7	9.18	76	
Skin	3	5.32	56	
All Other	34	23.28	146	.026
3. DISEASES OF THE CIRCULATORY SYSTEM	438	541.45	81	
Ischaemic Heart Disease	322	417.16	77	
Cerebrovascular Disease	52	57.87	90	
Other	64	66.42	96	
4. DISEASES OF THE RESPIRATORY SYSTEM	63	55.01	115	
Chronic Interstitial Pneumonia	15	2.49	602	.000
Influenza, Pneumonia, Bronchitis, and Asthma	32	40.92	78	
Other	16	11.60	138	
ACCIDENTS, POISONING, AND VIOLENCE	415	204.48	203	.000

Note:1. Some numbers do not sum due to rounding
2. Not all causes of death are shown

TABLE 5

MORTALITY DATA
COHORT 2: ALL EXCLUDING PYRS AFTER MILL (NOMILL)

CAUSE OF DEATH	OBSERVED	EXPECTED	SMR	P-VALUE
ALL CAUSES	1343	1205.24	111	.000
ALL DISEASE CAUSES	929	1002.46	93	
1. INFECTIVE DISEASES	9	7.26	124	
Silicotuberculosis	0	0.08	-	
Pulmonary Tuberculosis	3	1.81	166	
Other Tuberculosis	2	0.77	260	.086
All Other Infective Diseases	4	4.60	87	
2. MALIGNANT NEOPLASMS	315	261.66	120	.001
DIGESTIVE ORGANS	75	79.92	94	
Stomach	20	17.74	113	
Intestine or Rectum	34	33.53	101	
Other Digestive	21	28.65	73	
RESPIRATORY SYSTEM	158	88.39	179	.000
Nose, Nasal Cavities, etc.	0	0.82	-	
Larynx	2	4.09	49	
Trachea, Bronchus, Lung	156	82.74	189	.000
Other	0	0.74	-	
LYMPHATIC AND HEMATOPOIETIC TISSUE	21	28.65	73	
OTHER SPECIFIC MALIGNANT NEOPLASMS	61	64.70	94	
Bone	1	1.29	77	
Kidney	5	6.84	73	
Bladder	1	6.01	17	
Brain	10	13.22	76	
Prostate	7	9.03	77	
Skin	3	5.27	57	
All Other	34	23.04	148	.022
3. DISEASES OF THE CIRCULATORY SYSTEM	434	535.24	81	
Ischaemic Heart Disease	319	412.46	77	
Cerebrovascular Disease	52	57.14	91	
Other	63	65.65	96	
4. DISEASES OF THE RESPIRATORY SYSTEM	62	54.32	114	
Chronic Interstitial Pneumonia	15	2.46	609	.000
Influenza, Pneumonia, Bronchitis, and Asthma	32	40.42	79	
Other	15	11.45	131	
ACCIDENTS, POISONING, AND VIOLENCE	414	202.79	204	.000

Note: 1. Some numbers do not sum due to rounding
2. Not all causes of death are shown

TABLE 6

MORTALITY DATA
COHORT 3: GOLD

CAUSE OF DEATH	OBSERVED	EXPECTED	SMR	P-VALUE
ALL CAUSES	738	658.48	112	.002
ALL DISEASE CAUSES	540	572.45	94	
1. INFECTIVE DISEASES	7	3.88	180	.089
Silicotuberculosis	0	0.06	-	
Pulmonary Tuberculosis	3	1.04	289	.043
Other Tuberculosis	1	0.43	234	
All Other Infective Diseases	3	2.36	127	
2. MALIGNANT NEOPLASMS	183	148.14	124	.004
DIGESTIVE ORGANS	47	46.28	102	
Stomach	14	10.35	135	
Intestine or Rectum	18	19.45	93	
Other Digestive	15	16.48	91	
RESPIRATORY SYSTEM	95	51.31	185	.000
Nose, Nasal Cavities, etc.	0	0.44	-	
Larynx	1	2.32	43	
Trachea, Bronchus, Lung	94	48.16	195	.000
Other	0	0 39	-	
LYMPHATIC AND HEMATOPOIETIC TISSUE	11	14.80	74	
OTHER SPECIFIC MALIGNANT NEOPLASMS	30	35.75	84	
Bone	0	0.64	-	
Kidney	5	3.82	131	
Bladder	0	3.74	-	
Brain	4	6.62	60	
Prostate	2	6.01	33	
Skin	3	2.52	119	
All Other	16	12.39	129	
3. DISEASES OF THE CIRCULATORY SYSTEM	240	312.45	76	
Ischaemic Heart Disease	180	240.37	75	
Cerebrovascular Disease	23	34.22	67	
Other	37	38.87	98	
4. DISEASES OF THE RESPIRATORY SYSTEM	38	32.38	117	
Chronic Interstitial Pneumonia	11	1.47	746	.000
Influenza, Pneumonia, Bronchitis, and Asthma	18	24.09	75	
Other	9	6.82	132	
ACCIDENTS, POISONING, AND VIOLENCE	198	86.02	230	.000

Note: 1. Some numbers do not sum due to rounding
2. Not all causes of death are shown

TABLE 7

MORTALITY DATA
COHORT 3A: GOLD PRIOR TO URANIUM (GOLDBE)[1]

CAUSE OF DEATH	OBSERVED	EXPECTED	SMR	P-VALUE
ALL CAUSES	699	639.09	109	.018
ALL DISEASE CAUSES	516	557.63	93	
1. INFECTIVE DISEASES	7	3.77	186	.077
Silicotuberculosis	0	0.06	-	
Pulmonary Tuberculosis	3	1.02	295	.040
Other Tuberculosis	1	0.42	241	
All Other Infective Diseases	3	2.28	132	
2. MALIGNANT NEOPLASMS	175	144.17	121	.010
DIGESTIVE ORGANS	45	45.13	100	
Stomach	14	10.10	139	
Intestine or Rectum	17	18.97	90	
Other Digestive	14	16.06	87	
RESPIRATORY SYSTEM	91	50.04	182	.000
Nose, Nasal Cavities, etc.	0	0.43	-	
Larynx	1	2.26	44	
Trachea, Bronchus, Lung	90	46.98	192	.000
Other	0	0.37	-	
LYMPHATIC AND HEMATOPOIETIC TISSUE	10	14.29	70	
OTHER SPECIFIC MALIGNANT NEOPLASMS	29	34.71	84	
Bone	0	0.62	-	
Kidney	4	3.72	108	
Bladder	0	3.67	-	
Brain	4	6.37	63	
Prostate	2	5.92	34	
Skin	3	2.41	124	
All Other	16	12.01	133	
3. DISEASES OF THE CIRCULATORY SYSTEM	232	304.97	76	
Ischaemic Heart Disease	174	234.58	74	
Cerebrovascular Disease	23	33.46	69	
Other	35	36.93	95	
4. DISEASES OF THE RESPIRATORY SYSTEM	36	31.65	114	
Chronic Interstitial Pneumonia	11	1.44	763	.000
Influenza, Pneumonia, Bronchitis, and Asthma	17	23.55	72	
Other	8	6.66	120	
ACCIDENTS, POISONING, AND VIOLENCE	183	81.47	225	.000

Note: 1. Uranium miners with previous gold mining experience
2. Some numbers do not sum due to rounding
3. Not all causes of death are shown

TABLE 8

MORTALITY DATA
COHORT 3B: COLD AFTER URANIUM

CAUSE OF DEATH	OBSERVED	EXPECTED	SMR	P-VALUE
ALL CAUSES	39	19.38	201	.000
ALL DISEASE CAUSES	24	14.82	162	.017
1. INFECTIVE DISEASES	0	0.11	-	
Silicotuberculosis	0	0.00	-	
Pulmonary Tuberculosis	0	0.02	-	
Other Tuberculosis	0	0.01	-	
All Other Infective Diseases	0	0.08	-	
2. MALIGNANT NEOPLASMS	8	3.96	202	.041
DIGESTIVE ORGANS	2	1.15	174	
Stomach	0	0.25	-	
Intestine or Rectum	1	0.48	209	
Other Digestive	1	0.42	238	
RESPIRATORY SYSTEM	4	1.27	316	.019
Nose, Nasal Cavities, etc.	0	0.01	-	
Larynx	0	0.06	-	
Trachea, Bronchus, Lung	4	1.18	339	.014
Other	0	0.01	-	
LYMPHATIC AND HEMATOPOIETIC TISSUE	1	0.51	196	
OTHER SPECIFIC MALIGNANT NEOPLASMS	1	1.04	96	
Bone	0	0.02	-	
Kidney	1	0.11	928	.011
Bladder	0	0.07	-	
Brain	0	0.25	-	
Prostate	0	0.09	-	
Skin	0	0.11	-	
All Other	0	0.39	-	
3. DISEASES OF THE CIRCULATORY SYSTEM	8	7.48	107	
Ischaemic Heart Disease	6	5.78	104	
Cerebrovascular Disease	0	0.76	-	
Other	2	0.94	213	
4. DISEASES OF THE RESPIRATORY SYSTEM	2	0.73	274	.076
Chronic Interstitial Pneumonia	0	0.03	-	
Influenza, Pneumonia, Bronchitis, and Asthma	1	0.54	185	
Other	1	0.16	639	.022
ACCIDENTS, POISONING, AND VIOLENCE	15	4.56	329	.000

Note: 1. Some numbers do not sum due to rounding
2. Not all causes of death are shown

TABLE 9

MORTALITY DATA
COHORT 4: NOGOLD

CAUSE OF DEATH	OBSERVED	EXPECTED	SMR	P-VALUE
ALL CAUSES	605	546.77	111	.013
ALL DISEASE CAUSES	389	430.00	90	
1. INFECTIVE DISEASES	2	3.38	59	
Silicotuberculosis	0	0.02	-	
Pulmonary Tuberculosis	0	0.77	-	
Other Tuberculosis	1	0.34	292	.094
All Other Infective Diseases	1	2.24	45	
2. MALIGNANT NEOPLASMS	132	113.53	116	.083
DIGESTIVE ORGANS	28	33.64	83	
Stomach	6	7.39	81	
Intestine or Rectum	16	14.08	114	
Other Digestive	6	12.17	49	
RESPIRATORY SYSTEM	63	37.08	170	.000
Nose, Nasal Cavities, etc.	0	0.38	-	
Larynx	1	1.77	56	
Trachea, Bronchus, Lung	62	34.58	179	.000
Other	0	0.36	-	
LYMPHATIC AND HEMATOPOIETIC TISSUE	10	13.87	72	
OTHER SPECIFIC MALIGNANT NEOPLASMS	31	28.95	107	
Bone	1	0.65	153	
Kidney	0	3.01	-	
Bladder	1	2.28	44	
Brain	6	6.60	91	
Prostate	5	3.02	165	
Skin	0	2.75	-	
All Other	17	10.65	169	.024
3. DISEASES OF THE CIRCULATORY SYSTEM	194	222.79	87	
Ischaemic Heart Disease	139	172.10	81	
Cerebrovascular Disease	29	22.92	127	
Other	26	27.78	94	
4. DISEASES OF THE RESPIRATORY SYSTEM	24	22.00	109	
Chronic Interstitial Pneumonia	4	0.99	405	.007
Influenza, Pneumonia, Bronchitis, and Asthma	14	16.33	86	
Other	6	4.63	130	
ACCIDENTS, POISONING, AND VIOLENCE	216	116.77	185	.000

Note: 1. Some numbers do not sum due to rounding
2. Not all causes of death are shown

TABLE 10

MORTALITY DATA
COHORT 5: NOGOLD EXCLUDING NICKEL/COPPER (NONICU)

CAUSE OF DEATH	OBSERVED	EXPECTED	SMR	P-VALUE
ALL CAUSES	438	421.94	104	
ALL DISEASE CAUSES	281	329.94	85	
1. INFECTIVE DISEASES	2	2.63	76	
Silicotuberculosis	0	0.02	-	
Pulmonary Tuberculosis	0	0.60	-	
Other Tuberculosis	1	0.27	377	.059
All Other Infective Diseases	1	1.74	57	
2. MALIGNANT NEOPLASMS	98	86.95	113	
DIGESTIVE ORGANS	22	25.74	85	
Stomach	5	5.67	88	
Intestine or Rectum	12	10.77	111	
Other Digestive	5	9.30	54	
RESPIRATORY SYSTEM	44	28.24	156	.003
Nose, Nasal Cavities, etc.	0	0.29	-	
Larynx	0	1.35	-	
Trachea, Bronchus, Lung	44	26.33	167	.001
Other	0	0.28	-	
LYMPHATIC AND HEMATOPOIETIC TISSUE	9	10.73	84	
OTHER SPECIFIC MALIGNANT NEOPLASMS	22	22.26	103	
Bone	1	0.51	195	
Kidney	0	2.30	-	
Bladder	1	1.75	57	
Brain	5	5.06	99	
Prostate	4	2.36	169	
Skin	0	2.11	-	
All Other	12	8.17	147	
3. DISEASES OF THE CIRCULATORY SYSTEM	136	170.78	80	
Ischaemic Heart Disease	96	131.63	73	
Cerebrovascular Disease	22	17.73	124	
Other	18	21.42	84	
4. DISEASES OF THE RESPIRATORY SYSTEM	24	17.00	141	.090
Chronic Interstitial Pneumonia	4	0.76	525	.002
Influenza, Pneumonia, Bronchitis, and Asthma	14	12.68	110	
Other	6	3.57	168	
ACCIDENTS, POISONING, AND VIOLENCE	157	92.00	171	.000

Note: 1. Some numbers do not sum due to rounding
2. Not all causes of death are shown

TABLE 11

MORTALITY DATA
COHORT 6: NOGOLD EXCLUDING IRON (NOIRON)

CAUSE OF DEATH	OBSERVED	EXPECTED	SMR	P-VALUE
ALL CAUSES	576	522.52	110	.020
ALL DISEASE CAUSES	373	410.70	91	
1. INFECTIVE DISEASES	2	3.23	62	
Silicotuberculosis	0	0.02	-	
Pulmonary Tuberculosis	0	0.74	-	
Other Tuberculosis	1	0.33	305	.087
All Other Infective Diseases	1	2.14	47	
2. MALIGNANT NEOPLASMS	129	108.36	119	.047
DIGESTIVE ORGANS	27	32.12	84	
Stomach	6	7.06	85	
Intestine or Rectum	16	13.44	119	
Other Digestive	5	11.61	43	
RESPIRATORY SYSTEM	61	35.36	173	.000
Nose, Nasal Cavities, etc.	0	0.36	-	
Larynx	1	1.69	59	
Trachea, Bronchus, Lung	50	32.97	182	.000
Other	0	0.34	-	
LYMPHATIC AND HEMATOPOIETIC TISSUE	10	13.24	76	
OTHER SPECIFIC MALIGNANT NEOPLASMS	31	27.65	112	
Bone	1	0.63	160	
Kidney	0	2.87	-	
Bladder	1	2.18	46	
Brain	6	6.29	95	
Prostate	5	2.92	171	
Skin	0	2.62	-	
All Other	18	10.16	177	.014
3. DISEASES OF THE CIRCULATORY SYSTEM	184	212.84	86	
Ischaemic Heart Disease	132	164.29	80	
Cerebrovascular Disease	29	21.97	132	
Other	23	26.58	87	
4. DISEASES OF THE RESPIRATORY SYSTEM	24	21.04	114	
Chronic Interstitial Pneumonia	4	0.95	422	.006
Influenza, Pneumonia, Bronchitis, and Asthma	14	15.70	89	
Other	6	4.44	135	
ACCIDENTS, POISONING, AND VIOLENCE	203	111.82	182	.000

Note: 1. Some numbers do not sum due to rounding
2. Not all causes of death are shown

TABLE 12

MORTALITY DATA
COHORT 7: NOCOLD EXCLUDING ANY "OTHER ORE" (NOANY)

CAUSE OF DEATH	OBSERVED	EXPECTED	SMR	P-VALUE
ALL CAUSES	339	317.78	107	
ALL DISEASE CAUSES	214	246.28	87	.040
1. INFECTIVE DISEASES	2	1.98	110	
Silicotuberculosis	0	0.02	-	
Pulmonary Tuberculosis	0	0.45	-	
Other Tuberculosis	1	0.20	506	.034
All Other Infective Diseases	1	1.32	76	
2. MALIGNANT NEOPLASMS	63	64.94	97	
DIGESTIVE ORGANS	15	19.17	78	
Stomach	0	4.23	-	
Intestine or Rectum	12	8.03	149	
Other Digestive	3	6.91	43	
RESPIRATORY SYSTEM	23	20.95	110	
Nose, Nasal Cavities, etc.	0	0.21	-	
Larynx	0	1.00	-	
Trachea, Bronchus, Lung	23	19.54	118	
Other	0	0.21	-	
LYMPHATIC AND HEMATOPOIETIC TISSUE	7	8.11	86	
OTHER SPECIFIC MALIGNANT NEOPLASMS	18	16.71	108	
Bone	0	0.39	-	
Kidney	0	1.70	-	
Bladder	0	1.31	-	
Brain	2	3.79	53	
Prostate	3	1.79	168	
Skin	0	1.60	-	
All Other	13	6.13	212	.006
3. DISEASES OF THE CIRCULATORY SYSTEM	115	127.19	90	
Ischaemic Heart Disease	79	97.82	81	
Cerebrovascular Disease	19	13.31	143	
Other	17	16.06	106	
4. DISEASES OF THE RESPIRATORY SYSTEM	13	12.79	102	
Chronic Interstitial Pneumonia	2	0.57	349	.041
Influenza, Pneumonia, Bronchitis, and Asthma	8	9.53	84	
Other	3	2.68	112	
ACCIDENTS, POISONING, AND VIOLENCE	125	71.50	175	.000

Note: 1. Some numbers do not sum due to rounding
2. Not all causes of death are shown

TABLE 13

MORTALITY DATA
COHORT 8: ALL EXCLUDING PYRS PRIOR TO MILL (MILL)

CAUSE OF DEATH	OBSERVED	EXPECTED	SMR	P-VALUE
ALL CAUSES	9	13.06	69	
ALL DISEASE CAUSES	8	11.37	70	
1. INFECTIVE DISEASES	0	0.07	-	
Silicotuberculosis	0	0.00	-	
Pulmonary Tuberculosis	0	0.02	-	
Other Tuberculosis	0	0.01	-	
All Other Infective Diseases	0	0.05	-	
2. MALIGNANT NEOPLASMS	2	2.94	68	
DIGESTIVE ORGANS	2	0.91	219	
Stomach	1	0.20	490	.036
Intestine or Rectum	1	0.39	259	
Other Digestive	0	1.32	-	
RESPIRATORY SYSTEM	0	1.01	-	
Nose, Nasal Cavities, etc.	0	0.01	-	
Larynx	0	0.04	-	
Trachea, Bronchus, Lung	0	0.95	-	
Other	0	0.01	-	
LYMPHATIC AND HEMATOPOIETIC TISSUE	0	0.29	-	
OTHER SPECIFIC MALIGNANT NEOPLASMS	0	0.72	-	
Bone	0	0.01	-	
Kidney	0	0.07	-	
Bladder	0	0.08	-	
Brain	0	0.12	-	
Prostate	0	0.14	-	
Skin	0	0.05	-	
All Other	0	0.24	-	
3. DISEASES OF THE CIRCULATORY SYSTEM	4	6.20	64	
Ischaemic Heart Disease	3	4.70	64	
Cerebrovascular Disease	0	0.74	-	
Other	1	0.77	130	
4. DISEASES OF THE RESPIRATORY SYSTEM	1	0.69	145	
Chronic Interstitial Pneumonia	0	0.03	-	
Influenza, Pneumonia, Bronchitis, and Asthma	0	0.51	-	
Other	1	0.15	665	.020
ACCIDENTS, POISONING, AND VIOLENCE	1	1.69	59	

Note: 1. Some numbers do not sum due to rounding
2. Not all causes of death are shown

TABLE 14

LUNG CANCER: MORTALITY RISK BY CUMULATIVE WLM FOR THREE COHORTS: ANY, NOANY AND NOGOLD

CUMULATIVE WLM	ANY		NOANY		NOGOLD	
	OBS	SMR	OBS	SMR	OBS	SMR
0	0	-	2	132	2	94
1-19	10	160	8	89	18	118
20-39	6	213	5	133	11	167
40-59	8	567	0	-	8	279
60-79	4	404	3	294	7	350
80-99	1	106	1	159	2	128
100-149	3	375	0	-	3	160
150-199	3	476	1	172	4	331
200+	4	659	3	536	7	600
TOTAL	39	259	23	118	62	179

TABLE 15

LUNG CANCER: MORTALITY RISK BY AGE AT DEATH
FOR GOLDBE AND NOGOLDBE COHORTS

AGE AT DEATH	GOLDBE		NOGOLDBE	
	OBS	SMR	OBS	SMR
Under 55	22	146	35	195
55-64	39	199	26	203
65+	29	236	5	101
TOTAL	90	192	66	185

TABLE 16

GOLDBE COHORT: LUNG AND STOMACH CANCER MORTALITY RISK
BY DURATION OF COLD DUST EXPOSURE

DURATION OF EXPOSURE (IN YEARS)	LUNG CANCER			STOMACH CANCER		
	OBS	EXP	SMR	OBS	EXP	SMR
<5	34	20.60	165	7	4.36	161
5-9	19	12.18	156	2	2.57	78
10-14	21	6.70	314	4	1.46	273
15-19	8	4.71	170	1	1.04	96
20-24	4	1.88	213	0	0.45	-
25+	4	0.91	441	0	0.22	-
TOTAL	90	47.0	192	14	10.1	139

TABLE 17

COLDBE COHORT: LUNG AND STOMACH CANCER MORTALITY RISK
BY DURATION OF COLD DUST EXPOSURE PRIOR TO 1945

COLD DUST EXPOSURE	LUNG CANCER OBS	LUNG CANCER SMR	STOMACH CANCER OBS	STOMACH CANCER SMR
<5	61	168	11	145
5-9	22	306	0	-
10-14	7	232	3	389
15-19	0	-	0	-
20-24	0	-	0	-
TOTAL	90	192	14	139

TABLE 18

COLDBE COHORT: LUNG AND STOMACH CANCER MORTALITY RISK
BY FIRST YEAR OF COLD DUST EXPOSURE

FIRST YEAR OF EXPOSURE	LUNG CANCER Obs	LUNG CANCER Exp	LUNG CANCER SMR	STOMACH CANCER Obs	STOMACH CANCER Exp	STOMACH CANCER SMR
<1936	24	9.24	260	3	2.24	134
1936-1944	36	17.5	206	4	3.66	109
1945-1949	14	9.39	149	3	1.88	159
1950+	16	10.9	147	4	2.31	173
TOTAL	90	47.0	192	14	10.1	139

TABLE 19

COLDBE COHORT: LUNG AND STOMACH CANCER MORTALITY RISK
BY WEIGHTED COLD DUST EXPOSURE

WEIGHTED GOLD DUST EXPOSURE	LUNG CANCER OBS	SMR	STOMACH CANCER OBS	SMR
0-9 dust years	33	160	8	185
10-19	21	192	2	89
20-29	15	227	1	70
30-39	8	208	0	-
40-59	8	211	3	332
60+	5	397	0	-
TOTAL	90	192	14	139

TABLE 20

COLDBE COHORT: LUNG AND STOMACH CANCER MORTALITY RISK
BY TIME SINCE FIRST COLD DUST EXPOSURE

TIME SINCE FIRST EXPOSURE (IN YEARS)	LUNG CANCER Obs	Exp	SMR	STOMACH CANCER Obs	Exp	SMR
<5	0	0.03	-	0	0.02	-
5-9	0	0.25	-	0	0.14	-
10-19	5	3.65	137	3	1.16	259
20-29	24	15.5	155	3	3.42	88
30+	61	27.6	221	8	5.37	149
TOTAL	90	47.0	192	14	10.1	139

TABLE 21

LUNG CANCER MORTALITY RISK BY CUMULATIVE WLM FOR GOLDBE AND NOGOLDBE COHORTS

CUMULATIVE WLM	NOGOLDBE OBS	NOGOLDBE SMR	GOLDBE OBS	GOLDBE SMR
0	2	94	4	246
1-19	20	129	25	147
20-39	11	160	16	166
40-59	9	288	9	146
60-79	7	332	6	167
80-99	2	125	9	353
100-149	4	207	9	286
150-199	4	323	5	309
200+	7	571	7	408
TOTAL	66	185	90	192

TABLE 22

NOCOLDBE COHORT: RELATIVE AND ATTRIBUTABLE RISK BY EXPOSURE[1]

EXPOSURE CATEGORY (WLM)	MEAN EXPOSURE[2]	PERSON YEARS	OBS	EXP	RR[3]	AR[4]
0-20	8.93	42,348	17	14.34	1.19	62.71
20-40	29.15	20,242	11	6.64	1.66	215.50
40-60	48.58	10,251	8	3.00	2.67	487.81
60-80	68.80	5,717	6	2.06	2.92	689.62
80-100	89.35	3,745	2	1.56	1.28	117.88
100-150	121.08	4,939	4	1.90	2.11	426.21
150-200	172.81	3,395	3	1.21	2.49	528.45
200+	243.10	2,416	7	1.22	5.75	2,394.14

Notes:

1. The first 5 years of follow-up are excluded from all calculations in this table.

2. Mean Exposure weighted by Person Years.

3. RR = (Obs/Exp).

4. AR = [(Obs - Exp)/Person Years] x 10^6.

5. Relative Risk coefficient (intercept of unity): 1.76% per WLM.

6. Attributable Risk coefficient (zero intercept): 6.58 per WLM per 10^6 person years.

TABLE 23

GOLDBE COHORT: RELATIVE AND ATTRIBUTABLE RISK BY EXPOSURE[1]

EXPOSURE CATEGORY (WLM)	MEAN EXPOSURE[2]	PERSON YEARS	OBS	EXP	RR[3]	AR[4]
0-10	4.93	13,793	11	8.86	1.24	155.24
10-20	14.79	11,111	10	6.92	1.44	276.93
20-30	24.70	8,192	7	4.66	1.50	285.58
30-40	34.88	6,395	6	4.43	1.35	245.28
40-60	49.37	8,309	9	5.85	1.54	379.44
60-80	69.20	4,770	6	3.44	1.74	536.00
80-100	89.20	3,849	9	2.46	3.66	1,698.76
100-150	119.74	4,193	9	3.03	2.97	1,422.74
150-200	172.67	2,373	5	1.58	3.16	1,439.84
200+	241.49	1,985	7	1.70	4.13	2,671.54

Notes:

1. The first 5 years of follow-up are excluded from all calculations in this table.

2. Mean Exposure weighted by Person Years.

3. RR = (Obs/Exp).

4. AR = [(Obs - Exp)/Person Years] x 10^6.

5. Relative Risk coefficient (intercept of unity): 1.63% per WLM.

6. Attributable Risk coefficient (zero intercept): 11.36 per WLM per 10^6 person years.

TABLE 24

GOLDBE COHORT: MODELS OF STANDARDIZED MORTALITY RATIOS FOR LUNG CANCER

MODEL	REDUCTION IN DEVIANCE	REDUCTION IN DEGREES OF FREEDOM
UNADJUSTED DATA:		
(1.1) $SMR = 1.352 + 0.0219W_2 + 0.00897W_3$	5.98	2
(1.2) $SMR = 1.292 + A_1 \times [W_2 + 0.409W_3]$	11.42	4
where: $A_1 = 0.00622$ for ages at risk under 55 $A_2 = 0.02016$ " " 55-64 $A_3 = 0.05198$ " " 65+		
ADJUSTED DATA:		
(2.1) $SMR = (1.03 + 0.008\ DST) \times (1.159 + 0.0182W_2 + 0.00785W_3)$	5.84	2
(2.2) $SMR = (1.03 + 0.008\ DST) \times (1.118 + A_1 \times [W_2 + 0.432W_3])$	9.50	4
where: $A_1 = 0.00680$ for ages at risk under 55 $A_2 = 0.01656$ " " 55-64 $A_3 = 0.03742$ " " 65+		
(3.1) $SMR = (2.59 - 0.032\ YFEP) \times (1.091 + 0.01734W_2 + 0.00788W_3)$	6.12	2
(3.2) $SMR = (2.59 - 0.032\ YFEP) \times (1.063 + A_1 \times [W_2 + 0.455W_3])$	8.03	4
where: $A_1 = 0.01014$ for ages at risk under 55 $A_2 = 0.01507$ " " 55-64 $A_3 = 0.02991$ " " 65+		
(4.1) $SMR = (0.39 + 0.031\ TFE) \times (1.048 + 0.01787W_2 + 0.003738W_3)$	5.54	2
(4.2) $SMR = (0.39 + 0.031\ TFE) \times (1.009 + A_1 \times [W_2 + 0.209W_3])$	7.50	4
where: $A_1 = 0.00901$ for ages at risk under 55 $A_2 = 0.01747$ " " 55-64 $A_3 = 0.0348$ " " 65+		

Notes:

1. Models are estimated using Poisson regression techniques

2. Notation:
   DST, TFE, YFEP defined in Table 1
   W_2 WLM exposure 10-14 years prior to observation or death
   W_3 " " 15+ years " " "

3. Adjusted data incorporate gold mining risk into estimate of expected deaths

TABLE 25

NOCOLDBE COHORT: MODELS OF STANDARDIZED MORTALITY RATIOS FOR LUNG CANCER

MODEL	REDUCTION IN DEVIANCE	REDUCTION IN DEGREES OF FREEDOM
(1) $SMR = 1.382 + 0.0364W_2 + 0.004087W_3$	8.59	2
(2) $SMR = 1.334 + 0.0191W_1^B + 0.0088W_2^B$	7.28	2
(3) $SMR = 1.351 + A_1 \times [W_1^B + 0.464W_2^B]$	7.75	3

where: $A_1 = 0.02280$ for ages at risk under 55

$A_2 = 0.01399$ " " 55+

Notes:

1. Models are estimated using Poisson regression techniques

2. Notation:

W_2	WLM exposure	10-14 years	prior to	observation	or death
W_3	" "	15+ years	"	"	"
W_1^B	" "	5-14 years	"	"	"
W_2^B	" "	15+ years	"	"	"

TABLE 26

GOLDBE COHORT: COMPARISON OF DEVIANCE FOR VARIOUS MODELS OF LUNG CANCER RISK

	MODELS FROM TABLE 24	MODELS $W_2 + W_3$[1]	BEIR-IV FACTOR[2]
1.	No Adjustment (Eq. 1.1 and 1.2)	65.58	60.136
2.	Cum. Wtd. Gold Dust Exp. (Eq. 2.1 and 2.2)	63.53	59.87
3.	Calendar Year of First Exp. (Eq. 3.1 and 3.2)	61.88	59.96
4.	Time Since First Exp. (Eq. 4.1 and 4.2)	63.27	61.31

Notes:

1. Indicates equations containing separate exposure variables: W_2 and W_3 (i.e. Eq. 1.1, 2.1, 3.1 and 4.1)

2. Indicates equations containing interaction variable consisting of categorical variable for age multiplied by a linear combination of W_2 and W_3 (i.e. Eq. 1.2, 2.2, 3.2, 4.2)

TABLE 27

GOLDBE COHORT: PEARSON CORRELATION COEFFICIENTS BETWEEN ESTIMATES OF SMRs FOR LUNG CANCER OBSERVED DEATHS USING DIFFERENT MODELS

	MODELS (TABLE 24)					
MODELS (TABLE 24)	1.1	2.2	3.1	3.2	4.1	4.2
2.1	1.000	0.846	0.929	0.818	0.927	0.865
2.2		1.000	0.853	0.956	0.791	0.942
3.1			1.000	0.920	0.967	0.935
3.2				1.000	0.842	0.963
4.1					1.000	0.920
4.2						1.000

TABLE 28

GOLDBE COHORT: MODELS OF STANDARDIZED MORTALITY RATIOS FOR LUNG CANCER USING INTERCEPT OF UNITY FOR URANIUM MINING EFFECT

MODEL

UNADJUSTED DATA:

(1.1) $SMR = 1 + 0.0253W_2 + 0.0192W_3$

(1.2) $SMR = 1 + A_1 \times [W_2 + 0.758W_3]$

where: $A_1 = 0.0141$ for ages at risk under 55
$A_2 = 0.0238$ " " 55-64
$A_3 = 0.0386$ " " 65+

ADJUSTED DATA:

(2.1) $SMR = (1 + 0.0085\ DST) \times (1 + 0.0206W_2 + 0.0131W_3)$

(2.2) $SMR = (1 + 0.0085\ DST) \times (1 + A_1 \times [W_2 + 0.635W_3])$

where: $A_1 = 0.0115$ for ages at risk under 55
$A_2 = 0.0195$ " " 55-64
$A_3 = 0.0304$ " " 65+

(3.1) $SMR = (2.59 - 0.032\ YFEP) \times (1 + 0.0188W_2 + 0.0103W_3)$

(3.2) $SMR = (2.59 - 0.032\ YFEP) \times (1 + A_1 \times [W_2 + 0.547W_3])$

where: $A_1 = 0.0140$ for ages at risk under 55
$A_2 = 0.0171$ " " 55-64
$A_3 = 0.0249$ " " 65+

(4.1) $SMR = (0.39 + 0.031\ TFE) \times (1 + 0.0154W_2 + 0.00615W_3)$

(4.2) $SMR = (0.39 + 0.031\ TFE) \times (1 + A_1 \times [W_2 + 0.399W_3])$

where: $A_1 = 0.0107$ for ages at risk under 55
$A_2 = 0.0137$ " " 55-64
$A_3 = 0.0219$ " " 65+

Notes:

1. All equations were estimated using (SMR-1) as the dependent variable. Thus, no intercept models against which reductions in deviance could be measured were produced.

2. In equations (2.1) through (4.2), the SMR estimates were first adjusted for the indicated gold mining exposure effect before the Poisson regression procedure was applied.

3. All models are estimated using Poisson regression techniques.

TABLE 29

COMBINED COHORTS: MODELS OF STANDARDIZED MORTALITY RATIOS FOR LUNG CANCER

MODEL	REDUCTION IN DEVIANCE	REDUCTION IN DEG. OF FREEDOM
(1) $SMR = 1.386 + 0.0242W_2 + 0.00658W_3$	12.74	2
(2) $SMR = 1.304 + 0.0243W_2 + 0.00615W_3 + 0.0126DST$	14.21	3
(3) $SMR = 1.338 + 0.0242W_2 + 0.00656W_3 + 0.00447TFE$	13.17	3
(4) $SMR = 1.337 + 0.0242W_2 + 0.00672W_3 + 0.00342YFE$	13.38	3
(5) $SMR = 1.340 + A1\ [W_1^B + 0.5W_2^B]$ where: $A_1 = 0.01295$ for ages at risk under 55; $A_2 = 0.01626$ " " 55-64; $A_3 = 0.02668$ " " 65+	12.55	3
(6) $SMR = 1.305 + 0.0022TFE + A_1 \times [W_1^B + 0.5W_2^B]$ where: $A_1 = 0.0134$ for ages at risk under 55; $A_2 = 0.0160$ " " 55-64; $A_3 = 0.0257$ " " 65+	12.50	4
(7) $SMR = 1.323 + 0.00036\ YFE + A_1 \times [W_1^B + 0.5W_2^B]$ where: $A_1 = 0.0132$ for ages at risk under 55; $A_2 = 0.0166$ " " 55-64; $A_3 = 0.027$ " " 65+	13.25	4
(8) $SMR = 1.221 + 0.0119DST + A1 \times [W_2 + 0.411W_3]$ where: $A_1 = 0.0187$ for ages at risk under 55; $A_2 = 0.0265$ " " 55-64; $A_3 = 0.0254$ " " 65+	14.61	4
(9) $SMR = 1.315 + 0.0028YFE + A1 \times [W_2 + 0.328\ W_3]$ where: $A_1 = 0.0180$ for ages at risk under 55; $A_2 = 0.0269$ " " 55-64; $A_3 = 0.0300$ " " 65+	12.77	4
(10) $SMR = 1.337 + 0.0010\ TFE + A1 \times [W_2 + 0.375W_3]$ where: $A_1 = 0.0174$ for ages at risk under 55; $A_2 = 0.0261$ " " 55-64; $A_3 = 0.0302$ " " 65+	12.72	4

Notes:

1. YFE 1982 - Calendar Year of First Gold Gust Exposure;

   SMR, W_2, W_3, DST, TFE, W_1^B, W_2^B as before.

2. Models are estimated using Poisson regression techniques.

APPENDIX C

EVIDENTIARY SOURCE INFORMATION

ON

URANIUM MINING STUDY

APPENDIX C

EVIDENTIARY SOURCE INFORMATION ON URANIUM MINING STUDY

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APPENDIX D

THE ORIGINS OF RADIOBIOLOGICAL EFFECTS

IN

URANIUM MINING

APPENDIX D

THE ORIGINS OF RADIOBIOLOGICAL EFFECTS IN URANIUM MININC

1. INTRODUCTION

The material in this Appendix is drawn from the following sources: BEIR IV Committee Report, 1988; Cember, 1983; Cluff Lake, 1978; Coggle, 1983; Meredith, 1977; Thomas, 1982.

Everyone is exposed throughout their lives to ionizing radiation from both natural and artificial sources. The biological effects of ionizing radiation are caused by the absorption and distribution of the radiation energy in the tissues. If no energy were left behind in the tissue from the passage of radiation through living material, then there would be no biological effect. The different observed effects are related to the mass, charge and energy of the particular form of radiation.

2. PHYSICAL AND CHEMICAL PROPERTIES OF RADIATION

Matter is composed of elements, including 92 naturally occurring ones (e.g. helium, uranium, oxygen, etc.) plus several artificially produced. Elements are made up of atoms consisting of positively charged nuclei surrounded by negatively charged electron clouds. The nucleus contains protons (positively charged) and neutrons (electrically neutral). Protons and neutrons have the same mass and are about 1,800 times heavier than electrons. Atoms are usually electrically neutral, the number of protons in any nucleus equalling the number of electrons forming the cloud surrounding the nucleus. The atomic number of an element (A) equals the number of protons in its nucleus, while the mass number (M) is the number of protons and neutrons.

A given element (having a given atomic number) may have different isotopes (each defined by a different mass number). Thus, Table A following shows the decay products for the uranium series including $^{238}_{92}U$, a naturally occurring isotope (or nuclide) of uranium (atomic number 92) with mass number 238. About 99.3% of mined uranium is $^{238}_{92}U$, the other 0.7% being $^{235}_{92}U$. There are trace amounts of uranium-234 isotope as well. Uranium-238 and uranium-234 belong to one family, the uranium series shown in Table A, while uranium-235 is the first member of another series, the actinium series (not shown).

Most nuclides are stable, while others, such as uranium, undergo radioactive decay to produce more stable elements. Each 'radionuclide' decays in a characteristic way to produce one or more emissions of a defined energy. There are several different types of decay, including alpha, beta, gamma emissions, internal conversion and electron capture.

When a nucleus emits an alpha particle, its atomic number falls by 2 and its mass number by 4. Thus, the decay of uranium-238 by alpha-emission produces an isotope of thorium:

$$^{238}_{92}U \longrightarrow {}^{234}_{90}Th + {}^{4}_{2}He$$

Uranium-238 --> Thorium-234 + alpha particle

The alpha-particle (a helium nucleus) has an energy of about 4.18MeV (The electron volt (eV) is a measure of energy = 1.6×10^{-19} joules; 1 MeV = 10^6 eV). Most alpha-emitters are elements of high atomic number and the energy of alpha particles ranges from 4 to 9 MeV.

Beta particles are 'electrons', and are emitted from the nucleus by the conversion of a neutron into a proton (causing the atomic number to increase by one and the mass number to remain the same). Thus, in Table A, bismuth-210 decays by beta emission to produce polonium-210:

$$^{210}_{83}Bi \longrightarrow {}^{210}_{84}Po + {}^{0}_{-1}e$$

Bismuth-210 --> Polonium-210 + beta particle

Note that the beta particle has atomic and mass numbers of -1 and 0 respectively. After an alpha or beta emission, the nucleus may be in an excited state and will stabilize itself by releasing the excitation energy in the form of gamma rays. No change in atomic or mass number takes place with the release of gamma rays alone, since they are shortwave electromagnetic radiation. However, with one known exception (sodium-22), gamma radiation accompanies a particulate emission (e.g. an alpha particle) which in turn does change the atomic structure. Thus, in Table A, polonium-210 decays by alpha-emission and gamma-emission to lead-206, a stable element:

$$^{210}_{84}Po \longrightarrow {}^{206}_{82}Pb + {}^{4}_{2}He + \text{gamma}$$

Polonium-210 --> Lead-206 + alpha particle + gamma rays

Two other forms of radioactive decay also occur: internal conversion (by which gamma ray energy is transferred to an orbital electron of the element; this electron is subsequently ejected resulting in the release of characteristic X-rays as the remaining electrons adjust to fill the vacancy created by the ejected electron); and electron capture (whereby a proton in the nucleus captures an orbital electron, and is thereby transformed into a neutron with the emission of a neutrino; characteristic X-rays are emitted as the remaining electrons rearrange themselves). These types of radiation do not, however, occur in the uranium or thorium decay chains.

Radioactive decay takes place at random but always follows a pattern of exponential decay. A widely used measure of radioactive decay is the 'half-life' which denotes the time for the decay activity to reach one-half of its original value.

There are four chains of successive radioisotopes, three of which occur naturally (the uranium, actinium and thoron series), and one of which is artificially produced (the neptunium series). All three natural chains share several characteristics: the first member of each chain is long-lived (with a half life measured in geological time); all chains include a gaseous member, and the radioactive gas in each case is a different isotope of the element radon (the uranium series produces the gas $^{222}_{86}Rn$, called radon; the thoron series produces $^{220}_{86}Rn$, called thoron; the actinium series produces $^{219}_{86}Rn$, called actinon); the end product in all three series is lead. The existence of radioactive gasses in the three chains is one of the chief reasons for the presence of naturally occurring environmental radioactivity. The radon gas diffuses out of the earth into the air, and the radioactive radon daughters, which are solids under ordinary conditions, attach themselves to atmospheric dust. Table A shows the radioactive decay chain for the uranium series, which includes 'radon gas' ($^{222}_{86}Em$ or radon-222) and its 'daughters' or decay products. These short-lived radon daughters include: Polonium-218 ($^{218}_{84}Po$ or Radium A); Lead-214 ($^{214}_{82}Pb$ or Radium B); Bismuth-214 ($^{214}_{83}Bi$ or Radium C); and Polonium-214 ($^{214}_{84}Po$ or Radium C').

"Radon-222 is the most common isotope. Other radioisotopes of radon - radon-219 (actinon) and radon-220 (thoron) - occur naturally and have alpha-emitting decay products. Actinon has an extremely short half-life (3.9 s). Accordingly, concentrations of actinon and its daughters are extremely low, and decay of actinon contributes little to human exposure. Because of its short half-life (56 s), the concentration of thoron is also usually low. Dosimetric considerations suggest that the dose to the tracheobronchial epithelium from thoron progeny is, for an equal concentration of inhaled alpha energy, less by a factor of 3 than that due to the progeny of radon-222. The potential for lung cancer due to inhalation of thoron cannot be addressed directly, because the available epidemiological data are based almost exclusively on exposures to radon-222 and its daughters." (BEIR IV, 1988)

TABLE A

URANIUM SERIES (4n+2)

Nuclide	Half-Life	Energy in MeV -------- Alpha[1]	Beta	Gamma[2]
$^{238}_{92}U$	4.51×10^{9}y	4.18		
$^{234}_{90}Th(UX_1)$	24.10d		0.193, 0.103	0.092, 0.063
$^{234}_{91}Pa(UX_2)$	1.175m		2.31	1.0, 0.76
$^{234}_{91}Pa(UZ)$	6.66h		0.5	Many weak
$^{234}_{92}U(UII)$	2.48×10^{5}y	4.763		
$^{230}_{90}Th(I_0)$	8.0×10^{4}y	4.685		0.068
$^{226}_{88}Ra$	1,622y	4.777		
$^{222}_{86}Em(Rn)$	3.825d	5.486		0.51
$^{218}_{84}Po(RaA)$	3.05m	5.998[3]	Not known[3]	0.186
$^{218}_{85}At(RaA')$	2s	6.63[3]	Not known[3]	
$^{218}_{86}Em(RaA'')$	0.019s	7.127		
$^{214}_{82}Pb(RaB)$	26.8m		0.65	0.352, 0.295, 0.242
$^{214}_{83}Bi(RaC)$	19.7m	5.505[3]	1.65, 3.7[3]	0.609, 1.12
$^{214}_{84}Po(RaC')$	1.64×10^{-4}s	7.680		
$^{210}_{81}Tl(RaC'')$	1.32m		1.96	2.36, 0.783, 0.297
$^{210}_{82}Pb(RaD)$	19.4y		0.017	0.0467
$^{210}_{83}Bi(RaE)$	5.00d		1.17	
$^{210}_{84}Po(RaF)$	138.40d	5.298		0.802
$^{206}_{82}Pb(RaG)$	Stable			

Notes:

1 Only the highest energy alpha is given.

2 Only the most prominent gamma photons are listed.

3 Further decay proceeds by branching.

3. BIOLOGICAL EFFECTS FROM THE ABSORPTION OF IONIZING RADIATION

In order to affect matter, either living or non-living, radiation must deposit energy in its passage through the matter. The dissipation of particle radiation (from electrons, protons, neutrons, alpha particles, etc.) passing through matter results in the production of 'ion pairs'. The ionizations produced by non-particulate radiation (X-rays, gamma rays, etc.) occur indirectly. However, ionization events are held to be the principal cause of radiation effects in living matter. The exact mechanism for biological damage is not well understood.

The types of radiobiological damage that may occur include:

Molecular	Damage to enzymes, RNA and DNA, and interference with metabolic pathways;
Subcellular	Damage to cell membranes, nucleus, chromosomes, mitochondria and lysosomes;
Cellular	Inhibition of cell division; cell death; transformation to a malignant state;
Tissue; organ	Disruption of such systems as the central nervous system, the bone marrow and intestinal tract may lead to the death of animals; induction of cancer;
Whole animal	Death; 'radiation life shortening';
Populations of animals	Changes in genetic characteristics due to gene and chromosomal mutations in individual members of species.

The quantity and quality of the biological damage depend upon radiation dose, dose rate, and on the distribution of the dose in the tissues.

4. RADIATION DOSE

Until 1975, two units of radiation dosimetry were in common use: the Roentgen (R) and the rad.

The Roentgen is a unit of exposure, defined as the amount of radiation aimed at a target material, and not the fraction absorbed. It is defined as the quantity of X-ray or gamma radiation (below 2 MeV) such that the associated secondary electrons emitted (per cubic centimeter of air at 0 degrees C and normal pressure) produce ions of either sign carrying a charge of 2.58×10^{-4} coulombs per kilogram of air.

The Roentgen is a measure of the ability of a radiation beam to produce ionization in air, rather than a measure of the energy deposited in any actual amount of material. This distinction between exposure and deposited energy is exacerbated by several difficulties. Whereas, at low energies (<2 MeV), the ionization

produced by radiation occurs very close to the path of the ray itself, at higher energies both the ionization and the absorbed energy are more spread out and the measure of ionization is probably not a measure of total energy deposited. Also, for a given measure of ionization in air, the amount deposited in materials varies with the density and absorption properties of the material. Thus 1 R over a wide energy range deposits 88 ergs per gram in air, about 95 ergs in soft tissue and up to 4 times as much in bone. Lastly, by definition, the roentgen is only useable for x-ray and gamma radiation, and not for alpha rays or neutrons.

For these reasons, the International Commission on Radiological Units (ICRU), later called the International Commission on Radiological Protection (ICRP) in 1956 decided to adopt the rad (for Radiation Absorbed Dose) for radiobiological purposes. The rad is a measure of the radiation energy actually absorbed by tissues; and is defined as the absorption of 0.01 joules of radiation energy per kilogram of material (i.e. 1 rad = 0.01 J kg^{-1}). Since the energy absorbed by soft tissue from exposure to one roentgen is 0.0095 joules per kilogram, in practice these quantities (1 roentgen for 0.95 rads) have become interchangeable.

In the early 1970s, by international agreement, the Integrated System of Units (SI) began to be introduced into medical physics by the ICRU. The new SI unit of absorbed dose is the gray (Gy) defined as the energy absorption of 1 joule per kilogram (1 J kg^{-1}) which is one hundred times larger than the rad (1 Gy = 100 rad).

5. DOSE RATE

The rate at which a radiation dose is delivered to living material (in, for example, grays per hour or Gy h^{-1}) alters the biological effect produced. It is often observed that at low dose rates a smaller biological response is observed than at higher dose rates, suggesting that some repair of radiation damage is possible during irradiation at such low dose rates. However, other biological effects are not affected by dose rate, suggesting that no modification of the amount of certain kinds of radiation damage is possible.

6. DOSE DISTRIBUTION

The use of absorbed dose widened the scope of radiation dosimetry to include both alpha and beta radiation. But it also introduced another complication: the deposition of energy by different types of radiations does not generally lead to the same biological effect.

Different types of radiation vary in their effectiveness in damaging a biological system. To account for this fact, a measure

called the relative biological effectiveness (RBE) of a given type of radiation has been coined. The RBE compares the absorbed dose produced by a given radiation type with the dose from a standard type of radiation (say, x-rays or gamma rays) necessary to produce the same biological effect.

The RBE for a type of radiation depends on the distribution of ionizations and excitations it leaves in its tracks through material. The term linear energy transfer (LET), in keV per micrometer (or keV um^{-1}), is used to measure the mean energy released per unit distance traversed as the radiation type passes through living tissue. Alpha particles, neutrons and protons are high LET radiation while X-rays, gamma radiation and fast electrons are low LET radiations. High LET radiations are more damaging than low LET radiations; so that, for example, alpha particles have a higher RBE than do gamma rays. Generally, increasing LET leads to increasing RBE except for very high LET radiation where the RBE begins to fall off (i.e. because of the enormous energy, more of the energy is 'wasted').

To relate the notion of RBE to radiation of different types and differing values of LET, the term Quality Factor, Q, has been introduced by the ICRP as a multiplier of the physically absorbed dose to give a unit which is one of biologically equivalent dose. The concept of 'dose equivalence' makes it possible to determine the contributions by different types of radiation with different energy (i.e. LET) to the overall observed biological effect. The sievert (Sv) is the unit of dose equivalence in SI units and is numerically equal to a dose in grays multiplied by the quality factor (Q) of the radiation. The ICRP has produced quality factors for differing types of radiation with different LETs based to some extent on experiment. The old unit of dose equivalence was the rem, and 1 sievert = 100 rem.

7. URANIUM MINERS

The unit of activity of a radioactive material used to be the curie (Ci) defined as the number of disintegrations occurring in 1 gram of radium in equilibrium with its products which amounts to 3.7×10^{10} disintegrations per second. The corresponding SI unit is the becquerel (Bq) defined as one disintegration per second. Thus, one curie = 3.7×10^{10} Bq = 37 GBq (gigabecquerel).

The dose received by uranium miners involves internal irradiation from the inhalation of radon and its daughters and external whole body radiation. Measuring the absorbed dose is complicated both by the nature of alpha emissions (i.e. the short-lived progeny of radon and thoron daughters) as short range and high LET particles inside the body and by the determination of dose equivalance. It was not recognized until 1951 that the radon daughters trapped in the lungs were the chief causes of dose, because the radon itself,

an inert gas, is breathed in and out, while the short-lived daughters may be trapped in the lungs and may be built up over times of the order of their half-lives.

From this realization came the idea of measuring the energy released from the short-lived radon daughters and from this the unit called the Working Level: A person is said to be exposed to 1 Working Level (1 WL) if any combination of the short-lived daughters of radon in one liter of the surrounding air will result in the ultimate emission of 1.3×10^5 MeV of alpha particle energy. This is approximately the amount of alpha energy emitted by the short half-life daughters in equilibrium with 100 pCi of radon.

The WL is an exposure rate and is related to a dose equivalent rate. The total corresponding exposure is the sum of terms of appropriate WL multiplied by the time spent at each WL. The unit is the Working Level Month (WLM) which is the exposure during 170 working hours in a radon daughter concentration of 1 working level (WL). Therefore, 1 WLM corresponds to an equilibrium concentration of 1.7×10^4 pCi per litre of air (or 630 Bq l^{-1}).

Radon and thoron daughters are often attached to aerosol particles in the atmosphere and the deposition of these particles in the respiratory system of miners is very complex. The particles can reach deep into the lung whereas the more soluble, unattached radon and thoron daughters are deposited in upper airways. The dose to the lung is therefore very inhomogeneous and is influenced by the rate, depth and route (nose or mouth) of respiration, the geometry of the airways and the clearance and translocation patterns in the lungs. A WLM delivers about 10 mGy of alpha radiation to the bronchial epithelium and an estimated 5 mGy to the whole lung.

The January, 1978 amendments to the Atomic Energy Control Regulations established maximum permissible exposures to radon daughters for atomic radiation workers (including uranium miners). These levels of exposure are 2 WLM per quarter of a year with a maximum of 4 WLM for any year. In order to extend the regulation to all significant and different forms of radiation to which a worker may be exposed, it has been proposed that the sum of the fractions of the maximum permissible dose for each form of radiation should be less than one in any given year.

$$(E_1/P_1) + (E_2/P_2) + \cdots \leq 1$$

where E_1 is actual exposure of each form of radiation and P_1 is maximum permissible dose of that form of radiation. For example, if the first subscript refers to pure radon daughter exposure, then E_1, would be the actual annual WLM dose while P_1 would be the maximum permissible dose (say, 4 WLM per year).

TABLE B
CONVERSION UNITS

Units[1]	Conversion Factors
Electron Volt (eV)	1 eV = 1.6×10^{-19} joules
Becquerel (SI)	1 disintegration/s = 2.7×10^{-11} Ci
Curie	3.7×10^{10} disintegrations/s = 3.7×10^{10} Bq
Gray (SI)	1 J/kg = 100 rad
Roentgen (R)	1 R = 0.95 rads (in soft tissue)
Rad	100 erg/g = 0.01 Gy
Rem	0.01 Sievert
Sievert (SI)	100 Rem

Note:

1. International Units are designated (SI).

APPENDIX E

COMPARISONS OF LOW LATENCY LUNG CANCER CASES (<10 YEARS) AMONG URANIUM MINERS IN HAM COMMISSION REPORT (TABLE C.4) AND COMPARABLE CASES IN REPORT OF THE SPECIAL PANEL

APPENDIX E

COMPARISONS OF LOW LATENCY LUNG CANCER CASES (<10 YEARS) AMONG URANIUM MINERS IN HAM COMMISSION REPORT (TABLE C.4) AND COMPARABLE CASES IN REPORT OF THE SPECIAL PANEL

1. 14 cases of lung cancer with a total of at least 10 WLM in cumulative exposure and with less than 10 years of latency (or elapsed time from entry to uranium mining and death) were reported in Table C.4 of the Report of the Royal Commission on the Health and Safety of Workers in Mines (the Ham Commission Report). These were case numbers 1, 8, 13, 21, 27, 28, 31, 35, 36, 47, 53, 55, 57 and 61 (see Heller, Jan.10, 1989).

2. A further 5 cases of lung cancer with less than 10 WLM in cumulative exposure and with less than 10 years of latency are shown in Table C.4 (Case numbers 64, 66, 70, 80 and 81).

3. There are 12 cases out of the 90 lung cancer cases in the GOLDBE cohort with latencies of <10 years: and 7 out of 66 cases in the NOGOLDBE cohort. When these cases are matched with corresponding cases in Table C.4 using year of birth, year of death, year of entry into uranium mining, etc., the following table results:

MATCHING OF LUNG CANCER CASES IN HAM COMMISSION AND SPECIAL PANEL REPORTS

	In Table C.4		In RSP only		
	>10 WLM	<10 WLM	>10 WLM	<10 WLM	TOTAL
In GOLDBE Cohort	7	1	2	2	12
In NOGOLDBE Coh.	5	1	0	1	7
Table C.4 only	2	3			
TOTAL	14	5			

4. 12 out of the 14 cases in Table C.4 with >10 WLM can be linked leaving 2 unlinked cases from Table C.4. However, there are 2 cases in the RSP with >10 WLM which cannot be linked with Table C.4. So there is an 86% correspondence (12/14= 86%) on cases with >10 WLM total exposures. And there are 2 cases in each file which cannot be matched.

5. On low (<10 WLM) exposure cases, the match includes 2 out of 5 cases for a 40% correspondence. However, there are 3 unmatched cases from Table C.4 and 3 unmatched from the RSP in this category.

6. **Conclusion:**

a) In the cases of most interest (higher exposure cases), there is a high correspondence (86%) and there are 2 unmatched cases in each file.

b) Although the degree of matching in the low exposure cases is low (40%), there are 3 unmatched cases in each file.

c) There are a total of 19 cases in each file with the overall degree of correspondence being 74% (14 out of 19 cases).

d) It is unlikely that the 5 unmatched cases in each file are the same men. Possible reasons for the continuing discrepancy include:

- The Ham Commission probably included miners with extra-Ontario uranium mining experience which the RSP would have excluded;

- The Mining Master File was still in a developmental phase when the Ham Commission Report was published so that the RSP is likely dealing with a more complete list of Ontario uranium miners.